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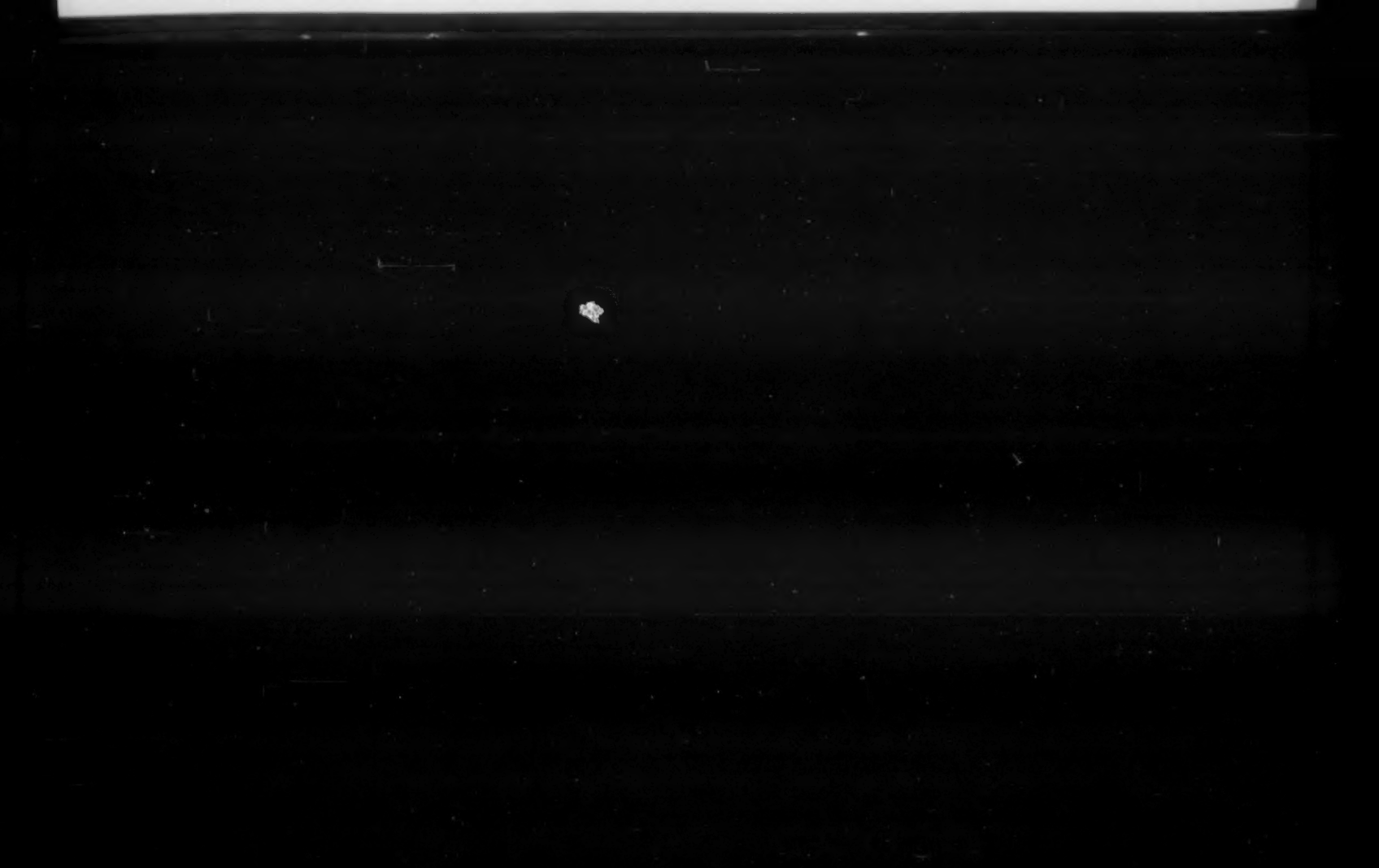

THE
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IMPORTANCE OF THE COLLAPSIBILITY OF THE ENDOLYMPHATIC LABYRINTH DURING FENESTRATION OPERATIONS.*

H. ENGSTROM, M.D.,
and
G. LIDÉN, M.D.,

with a Mathematical Analysis by
B. JAKOBSSON, Dr. Sc.,†
Göteborg, Sweden.

In the great number of otosclerosis operations performed during the last few decades, many surgeons have observed that the membranous labyrinth occasionally collapses when the fluid is aspirated from the field of operation. In 1951 Max Meyer published a work in which he considered related problems. He states in his work that when he aspirated the fluid in the neighborhood of the newly created fenestra, not only did perilymph around the membranous labyrinth disappear, but also the membranous labyrinth collapsed and lay "flat and completely invisible in contact with the bony wall of the labyrinthine perilymph space."

Based upon repeated operations and deliberate studies of the problem, Meyer published his work, in which he stated that "the membranous walls of the endolymphatic labyrinth are readily permeable from within outward, and also from

*From the Oto-laryngological Clinic of Sahlgrenska Sjukhuset, Göteborg. (Head: Prof. C. A. Hamberger, M.D.)
†Chalmers Technical University, Göteborg.
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without inward, for endolymph and perilymph and for many other liquids such as physiological saline solution, Ringer's solution, and low molecular dyes."

In 1954 Meyer's observations were brought up for discussion by Lempert, Wever and Lawrence, who in series of experiments had attempted to repeat Meyer's studies and to supplement these studies further with their own. In these experiments, they failed to find grounds for supporting Meyer's observations concerning the characteristics of the membranous labyrinth. They believed that the explanation of Meyer's observation depended upon an incomplete closure

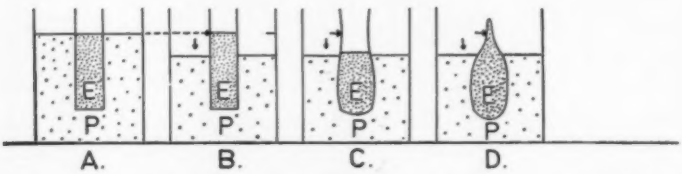

Fig. 1. Schematic drawing of different possibilities in the relation of endolymph (E) and perilymph (P) dependent on the characteristics of the endolabyrinthine wall or the characteristics of the container of a fluid.

A.—Rigid wall: E and P at the same level; B.—Rigid, closed container. With the P-level low, the E-level will be constant; C.—The wall of the container is elastic, and when the P-level is lowered, the E-level will also sink. This is accompanied by a certain distension of the E-wall; D.—If the E-container has an elastic wall and is totally filled with E-fluid of the same specific gravity as the P-fluid, a lowering of the P-level will result in a collapse of the upper part of the E-container and a distension of its lower part.

with subsequent leakage in the wall of the endolabyrinth. Instead, they assumed that the "collapse" of the labyrinth, also observed by them, depended upon an optical illusion.

Since, however, many surgeons have repeatedly, also observed a collapse of the endolabyrinth in connection with aspiration of perilymph, and since well-founded reason exists that such a collapse really occurs and is based upon basic physical principles, it is our intention to discuss this problem further.

OBSERVATIONS.

In a large number of otosclerosis operations, the authors

have repeatedly been able to observe a collapse of the endolabyrinth whenever surrounding perilymphatic fluid was aspirated. The extent of the collapse seems to depend upon the amount of aspirated perilymph. In certain model experiments we have shown that a collapse of a completely closed latex tube behaves in exactly the same manner as the endolabyrinth; and it will be shown that this depends upon simple physical principles which are capable of explaining Meyer's and Lempert's observations.

The endolabyrinth consists of a thin-walled and highly irregular formation which is completely separated from the perilymph. This more or less sac-formed endolymph container lies partially floating in the perilymph. At least in the region of the semicircular canals, it is suspended merely by thin connective tissue threads containing minute blood vessels; however, in those regions, where nerve fibers enter the sensory epithelia, the connection with the wall is considerably more firm. In a fenestration operation, the fistula is mainly created within that region of the semicircular canal where it is connected to the bony wall only by the strands mentioned above. It is in this region that a collapse can be observed during otosclerosis operations.

The condition necessary for a collapse of the semicircular canal is that a given amount of perilymph is aspirated. The semicircular canal collapses as soon as a certain difference of level occurs between the peri- and endolymph and only when the perilymph level is beneath that of the semicircular canal. Immediately following the aspiration of perilymph, the collapse may be observed. As soon as the perilymph is replaced (*e.g.* with physiological saline solution), the semicircular canal resumes its original form. Using thin pipettes, it is possible to aspirate and replace the fluid repeatedly; each time collapse or return to normal shape can be observed. It does not seem logical that osmotic or other permeability conditions could result in such a rapid change. Should the solution of the problem be merely an optical illusion, as Lempert, Wever and Lawrence have proposed, this phenomenon could quite easily be explained away.

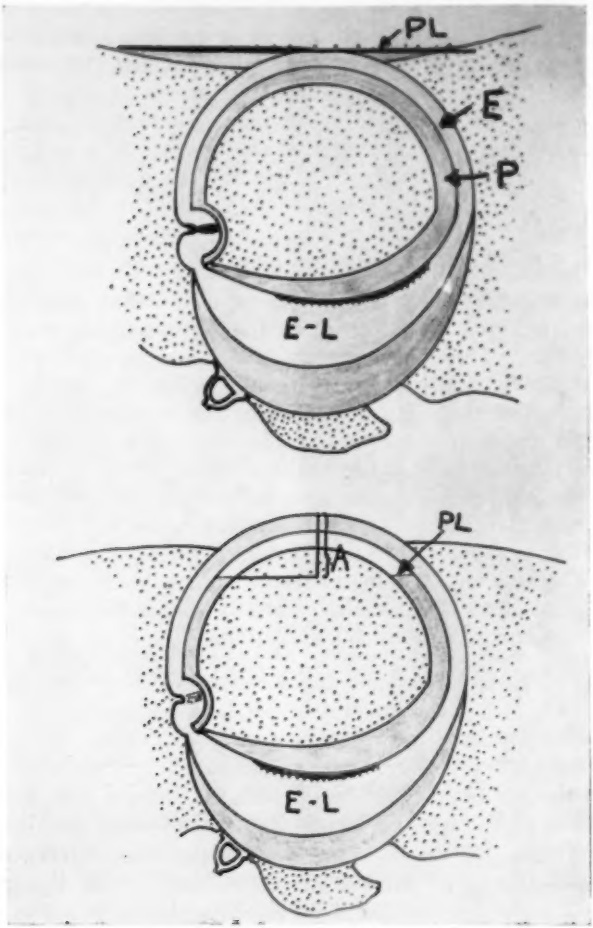

Fig. 2. Schematic drawings of the relation between endolymph (E) and perilymph (P), with two different positions of the perilymphatic level (PL).

Above: Perilymphatic level higher than the highest point of the endolabyrinth (E-L). Result: No collapse.

Below: Perilymphatic level lower (A) than the highest point of the semicircular canal. Result: The semicircular canal will collapse if it is not rigid.

If a simple latex model of the endolabyrinth is filled with a fluid of known specific weight, and submerged in a fluid of the same specific weight, it is easily demonstrated that an aspiration of the surrounding fluid results in a collapse of the wall of the model. This collapse occurs when the level of the surrounding fluid recedes below the highest point of the latex tube.

This experiment has been performed by the authors in the following manner: A circular latex tube was placed around a plastic center, filled with colored water, and submerged in a large glass filled with colorless water. If the surrounding water was higher than the highest point of the model, the latex tube was evenly filled by the colored water. Whenever the surrounding water was aspirated and the level sank beneath that of the uppermost point of the model, the soft latex tube began to collapse; the extent of this collapse was determined by the amount of fall of the surrounding fluid.

In principle, it appears that the collapse depends upon basic hydrodynamics and these principles are shown by Fig. 1, and also by the mathematical analysis in the appendix.*

It is our contention that the peri- and endolymph could be considered as two hydrodynamic systems with similar specific gravities, and that these two systems are separated by a thin resilient wall, the endolymphatic wall.

If a difference of level between the systems arises they attempt to establish a new equilibrium; *i.e.*, in this case, equal levels. This tendency is resisted by the stiffness of the endolymphatic wall; however, if this stiffness is slight, the wall gives way; consequently, the area which lies above the level of the "perilymph" will either wholly or partially collapse. An explanation according to the simple hydrodynamical principles mentioned above is capable of explaining the conditions observed by both researchers and surgeons.

Actually, however, the problem also has further clinical

*Appendix—Hydromechanical Treatment of the Problem—B. Jakobsson. The problem of the interaction between a fluid within a thin-walled container and a surrounding fluid can be given a simple analytical treatment.

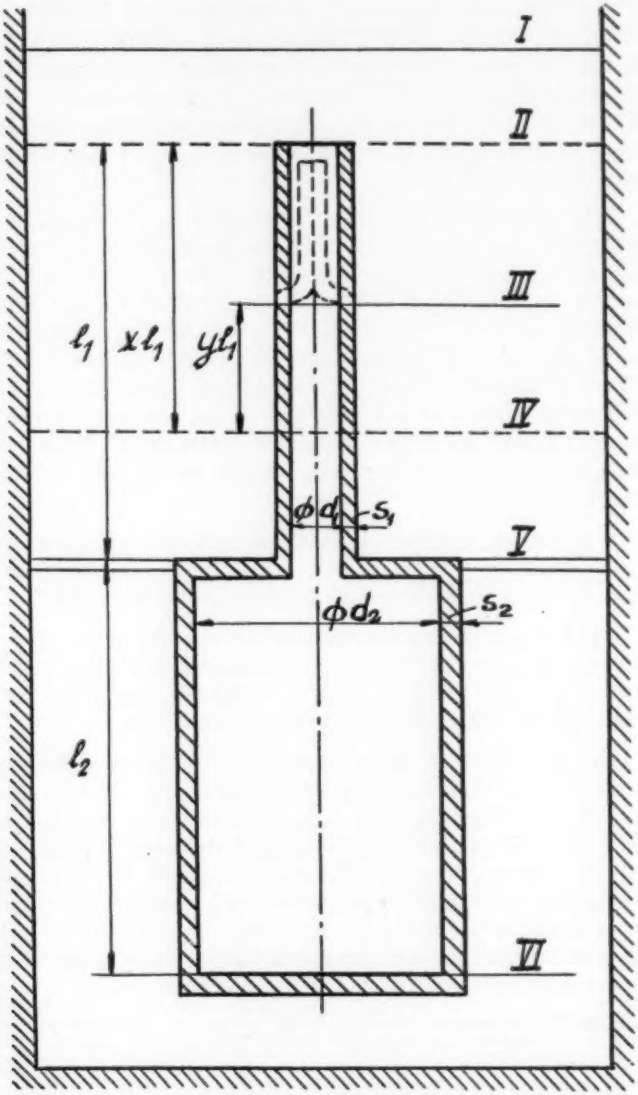

Fig. 3. See text.

interest. In connection with the collapse, the fluid which was inside the collapsed area must be displaced; this displacement should occur both "centrifugally" and "centripetally." If sufficient fluid is aspirated and this aspiration is rapid enough, the displacement of the inner fluid could influence the position and movement of the cupula; furthermore, a certain distention occurs within the endolabyrinth and should be able to change the functional conditions within the utricle and possibly also within the saccule. In some operations patients have reacted to the aspiration by nausea, vertigo and nystagmus. Thus it appears obvious, from theoretical as well as clinical experiments, that the surgeon should strive to maintain a level of fluid in the perilymphatic space which is not even momentarily allowed to drop below that of the endolymphatic labyrinth.

SUMMARY.

The authors discuss the problem of the collapse of the endolymphatic semicircular canal during the fenestration operation. Based upon studies made during fenestrations and model experiments, the authors conclude that the collapse depends upon simple hydrodynamical principles. This is further illustrated by a mathematical analysis. The clinical importance of the phenomenon is emphasized.

The container may consist of two thin-walled cylindrical parts as in Fig. 3. The initial condition is that the outer fluid level is at I while the container is completely filled with fluid. The two fluids are assumed to have the same specific gravity and to be incompressible. As the container wall is very weak the fluid pressure is equal on both sides of the container wall. The container is suspended in any way at section V. The dimensions shown in Fig. 3 refer to the initial state. The wall thickness is s.

Lowering the outer level to II gives rise to no changes in the form of the container. The decreasing hydrostatic pressure gives no volume change as the fluid is incompressible.

Lowering the outer level to IV, which is higher than V, will leave the inner fluid level at III. When the inner fluid level sinks, the atmospheric pressure will compress the top part of the weak walls of the container as shown dotted in Fig. 3.

The hydrostatic pressure on the container walls increases from $p_{III}=0$ at III to $p_{IV}=y_{l_1}\rho g$ (dyn/cm²) at IV where ρ is the density (g/cm³) and $g=981$. The rest of the container walls are expanded by the pressure p_{IV}. This pressure gives, after well known formulae (see standard books on Strength of Materials and Theory of Elasticity), an increase of the diameters d_1 and d_2 and the length l_2.

The diametral strain is:

$$\left.\begin{array}{ll}\text{from IV to V} & \epsilon_1=\dfrac{p_{IV}\ d_1}{2E\ s_1}\\ \text{from V to VI} & \epsilon_2=\dfrac{p_{IV}\ d_2}{2E\ s_2}\end{array}\right\}\ \text{N.B.}\qquad\qquad(1)$$

and the longitudinal strain:
from V to VI

$$\epsilon_l = \frac{\epsilon_2}{2}$$

where E is the modulus of elasticity (dyn/cm²).

The mean diametral strain is:

from III to IV $\frac{\epsilon_1}{2}$

With the section areas $A = \frac{\pi d^2}{4}$ consideration of the volume of the incompressible inner fluid initially and after lowering to III gives the equation:

$$l_1A_1 + l_2A_2 = y\,l_1\ A_1(1+2\cdot\frac{\epsilon_1}{2}) + (1-x)\,l_1A_1(1+2\epsilon_1) + l_2(1+\frac{\epsilon_2}{2})A_2(1+2\epsilon_2) \quad \ldots\ldots (2)$$

Here the deflection of the horizontal walls at V and VI as well as end effects of the cylindrical parts at V and VI are neglected.

With the symbol $m = \frac{l_2A_2}{l_1A_1} = \frac{V_2}{V_1}$ and neglecting the small quantity $\epsilon\frac{2}{2}$ eq. 2 gives:

$$x = \frac{y(1+\epsilon_1) + 2\epsilon_1 + 2.5m\epsilon_2}{1+2\epsilon_1} \quad \ldots\ldots (3)$$

Insertion of p_{1V} in (1) gives:

$$\left.\begin{aligned} \epsilon_1 &= y \cdot \frac{g\rho\, l_1 d_1}{2Es_1} \\ \epsilon_2 &= y \cdot \frac{g\rho\, l_1 d_1}{2Es_1} \cdot \frac{d_2}{d_1} \cdot \frac{s_1}{s_2} \end{aligned}\right\} \quad \ldots\ldots (4)$$

With the notation:

$$\epsilon_0 = \frac{g\rho\, l_1 d_1}{2Es_1} \qquad\qquad n = \frac{1}{2} \cdot \frac{d_2}{d_1} \cdot \frac{s_1}{s_2}$$

eqs (4) are:

$$\epsilon_1 = y\epsilon_0 \qquad\qquad \epsilon_2 = 2ny\epsilon_0$$

which inserted in eq (3) give:

$$x = y \cdot \frac{1+\epsilon_0(2+5mn+y)}{1+2y\epsilon_0} \quad \ldots\ldots (5)$$

Multiplying through by $(1-2y\epsilon_0)$ and neglecting terms with the small quantity $\epsilon\frac{2}{0}$ gives the following form of eq (5):

$$x = y\,[1+\epsilon_0(2+5mn-y)] \quad \ldots\ldots (6)$$

As $0 \leq x \leq 1$ and $0 \leq y \leq 1$ the parenthesis of eq (6) is > 1 and therefore, as shown in Fig. 3, $y < x$ and $0 \leq y \leq x$.

If to $x = 1$ corresponds the value $y = y_1$ eq (6) can be written:

$$x = \frac{y}{y_1}\left[1 + \frac{(1-y_1)\,(y_1-y)}{2+5mn-y_1}\right] \quad \ldots\ldots (7)$$

If the lower part of the container is large and not too thickwalled compared to the upper part, the parenthesis will be approximately equal to 1 which simply means linearity between x and y.

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SOME INTERESTING MIDDLE EAR PROBLEMS.*†

HAROLD F. SCHUKNECHT, M.D.,
and
SAMUEL TRUPIANO, M.D., (By Invitation),
Detroit, Mich.

The development of new surgical procedures has made it possible for present day otologists to explore, describe and correct an increasing number of middle ear disorders. Recently we have encountered several interesting middle ear conditions which have received little attention in the medical literature.

A. DISLOCATION OF THE INCUS.

The dislocation of the incus during simple mastoidectomy results in a hearing loss which is permanent unless reconstructive tympanic surgery is performed subsequently. The case history of a patient with a dislocated incus follows:

Case 1. R.B.—No. 809354—age 16, m.w., This boy had bilateral simple mastoidectomies performed elsewhere at the age of two-and-a-half years. When the otorrhea subsided he complained of hearing loss which subsequently created considerable handicap. At the age of 15 he acquired a hearing aid for the left ear and used it continuously.

We first examined him at the age of 16, when he stated that two months previously, following swimming, he experienced left earache followed by purulent discharge. When the discharge subsided one week later he noticed periodic improvement in hearing in that ear. When placing his head in the erect position he experienced a "clicking" sensation in the left ear, whereupon hearing improved greatly and remained at the improved level so long as his head remained erect (see Fig. 1-A. With flexion of the head the auditory acuity immediately dropped to its previous level. Over a period of six months it became progressively more difficult for him to maintain the improved hearing level. Seven months after onset he had lost the ability to acquire the better hearing state, and was again wearing the hearing aid.

Examination revealed depressed post-auricular scars indicating an extensive surgical exenteration of the mastoid. The external auditory canals were tortuous and reduced to half the normal diameter. The posterior halves of the tympanic membrane could be visualized and were

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*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Cleveland, O., Jan. 9, 1957.

†From the Division of Otolaryngology, Henry Ford Hospital, Detroit, Mich.

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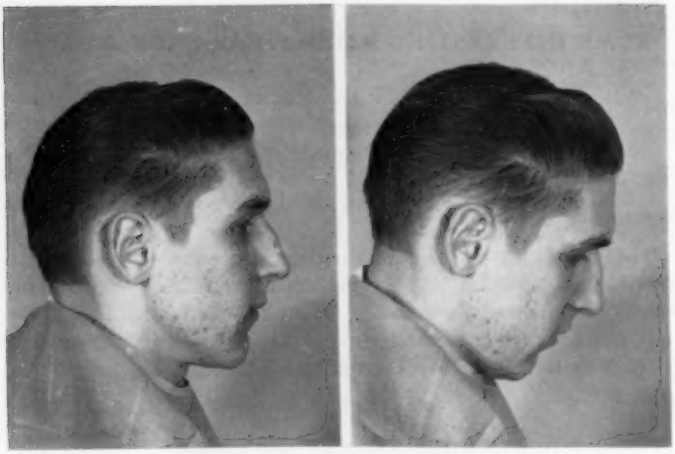
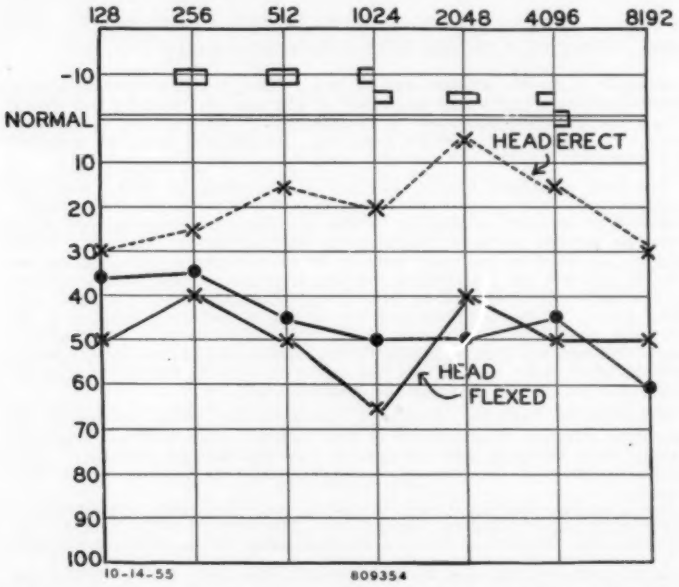


Fig. 1-A. Case 1. The hearing in the left ear was much better in the erect than in the flexed position of the head.



B. Audiogram showing improvement in thresholds in left ear when head was in erect position.

intact. Audiograms taken in both positions of the head are shown in Fig. 1-B.

An exploratory left trans-meatal operation was performed on March 14, 1956. Incisions were made to create a posterior tympano-meatal flap, which was elevated and reflected anteriorly to expose the middle ear cavity. The incus was found to be displaced downward so as to lie in the region between the handle of the malleus and the stapes (see Fig. 2-A). When the incus was rotated very slightly in a counter-clockwise direction, it made contact both with the head of the stapes and the handle of the malleus. The bone was readily forced upward into its normal position, whereupon the lenticular process appeared to make normal contact with the head of the stapes. Surgical audiometry revealed thresholds improved for low frequencies and worse for high frequencies (see following table):

SURGICAL AUDIOMETRY.

	500	1000	2000	4000	6000
*Before	40	40	35	25	20
After	25	25	35	35	50

For several weeks the thresholds remained near normal, but from the second month to the present time (nine months) there has been a slowly progressive loss; nevertheless, the hearing is still better than that prior to surgery (see Fig. 2-B.)

*The "before" thresholds were taken in all cases after middle ear exposure had been made and the tympano-meatal flap returned to normal position. The "after" thresholds were made after completion of surgery with the tympano-meatal flap again returned to normal position.

DISCUSSION.

Apparently the incus was dislocated at the time of simple mastoidectomy at the age of two-and-a-half years, and lay in the middle ear for 14 years. Normally the incus is fully developed at birth; therefore, it was possible in this case to return it to its normal position with almost perfect return of function. It seems likely that the incus was held in place for several weeks by soft tissue, and the subsequent decline in auditory function suggests that these tissues may have receded gradually from the incus and allowed it to slip out of firm contact with either, or both, the malleus and stapes. In this case the incus may be considered to be an autogenous dead bone transplant, and its failure to stay in position is not surprising. Possibly, a homologous fresh incus transplant would be more successful. The problem of interposing a functioning structure between the malleus and stapes is an intriguing one, beset with many difficulties. Some of the

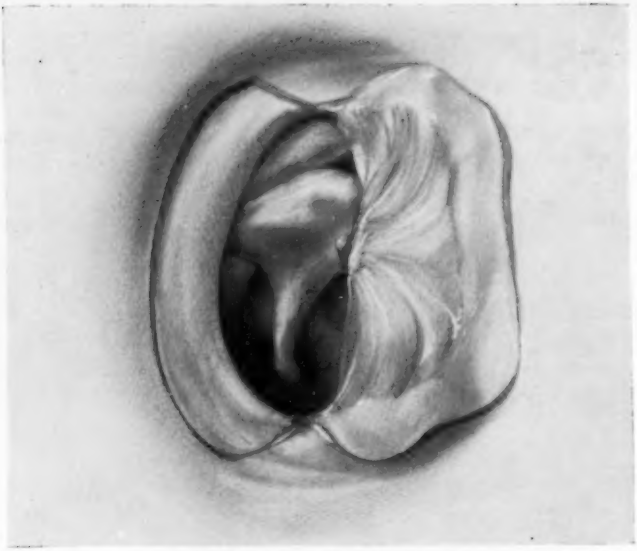
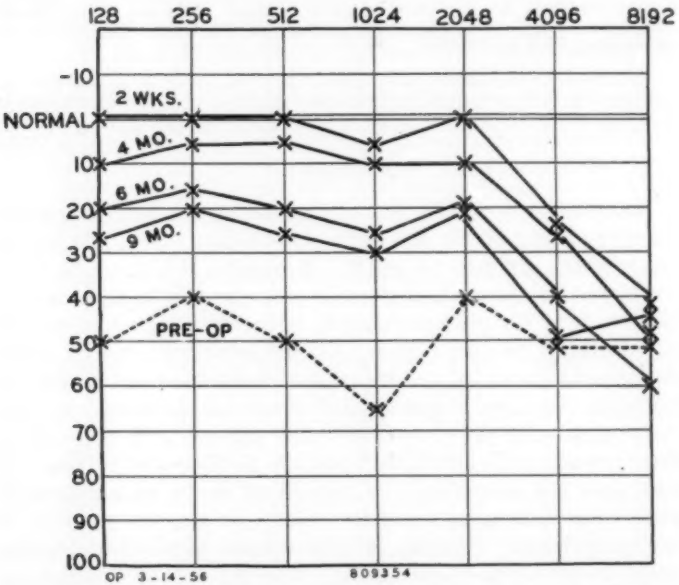


Fig. 2-A. Case 1. At surgical exploration the incus was found to be displaced down into the middle ear as shown in this drawing.



B. By placing the incus back into its normal position hearing was greatly improved for several weeks. Subsequently, however, there has been a slowly progressive loss although the thresholds are still better than pre-operative levels.

problems are: *1.* lack of a vascular bed; *2.* maintaining the position of either a living or artificial transplant, and *3.* avoiding ankylosis and stiffening of the reconstructed ossicular chain.

We are planning to perform a myringo-stapediopexy on this patient's opposite ear during his next school vacation. A satisfactory columella effect produced in this manner should result in auditory thresholds within 10-20 db. of normal.

B. JUDICIOUS USE OF THE HAMMER ON THE FOOTPLATE.

Since Rosen first demonstrated that it is possible to restore hearing in otosclerosis by mobilizing the footplate of the stapes bone, many new maneuvers and instruments have been advocated to increase the percentage of successful mobilizations. When mobilization is not readily accomplished by pressure exerted through the head, neck or crura, a variety of procedures are being applied to the footplate, including chiseling, needling, drilling and hammering. There is no doubt that the judicious use of these methods produces an increased percentage of good results, particularly if accomplished without crural fracture. The question arises, however, whether or not there is danger of creating traumatic acoustic impulses by these maneuvers. It is unlikely that drilling or needling will result in injury to the cochlea by acoustic stimulation, because we have used dental burrs and needles directly on the cochlear walls of many animals without creating stimulation deafness. A narrow, sharp chisel probably would create a less intense acoustic signal than a dull, broad one. Acoustic impulses of higher intensity would be expected to result from the blows of a blunt instrument on the footplate. The mechanism is the same as that resulting from a blow to the skull, for it is well established that stimulation deafness may follow a blow to the head. We have previously shown that animals, given an experimental head blow, may sustain high tone deafness and damage to the organ of Corti in the basal turn. The type of pathological change in the cochleae of these animals was exactly the same as that of animals subjected to high intensity blast or noise.[1]

In certain patients in whom the stapes footplate was firmly ankylosed we have resorted to the use of a blunt hammer of the type described by House. This pneumatic instrument is electrically driven and has several shapes of attachable blunt hammers which are specially adapted for the mobilization procedure.

We have used the hammer in 6 of 90 patients operated upon. In each case we had already fractured the crura and were unable to mobilize the footplate by needling its margins. The hammer blows resulted in the fracture of the footplate in four of the six cases. In five of the six patients surgical audiometery showed an increased hearing loss for 4000 CPS and 6000 CPS immediately after the hammer blow. The increased losses varied from 15 to 40 db in the different cases. In four of these five patients the bone conduction thresholds returned to their previous level within a few weeks of the operation, but one patient sustained a permanent loss of hearing.

The increased hearing losses were demonstrated by surgical audiometery performed immediately before and after the hammer blows, all conditions remaining unchanged. Elevation of bone conduction thresholds in the immediate postoperative period lends further support to the concept that these high frequency losses are due to inner ear damage. None of these six patients experienced significant hearing improvement. Because we have been unsuccessful with the hammer we have discontinued its use. The following are case histories of two patients on whom the hammer was used:

Case 2. W.G.—No. 842516—age 34, f.w. This patient had a progressive, bilateral hearing loss for 18 years. The tympanic membranes appeared normal, the Rinne test was negative bilaterally, and the audiogram revealed a bilateral conductive deafness.

On August 4, 1956, an operation was performed to mobilize the stapes. The footplate appeared to be thickened and was firmly fixed. After repeated attempts to mobilize it by forces applied to the capitulum and needling the margins of the footplate, the crura were fractured. The pneumatic hammer was then used on the footplate. Three series of blows were delivered, three blows in each series, making a total of nine blows. The footplate remained immobile. Surgical audiometry revealed a significantly increased loss for the 4000 CPS frequency, so no further attempts were made to mobilize the footplate.

SURGICAL AUDIOMETRY.

	500	1000	2000	4000	6000
Immediately before Hammer	65	35	45	60	Not heard at 80
Immedately after Hammer	65	60	50	Not heard at 60	Not heard at 80

When first tested three weeks after operation the loss for high frequencies was shown to involve both bone and air conduction thresholds. This loss was presumably the result of inner ear damage. When tested again five months later the bone conduction was back to the pre-operative level (see Fig. 3-A).

Case 3. J. A.—No. 822212—age 55, f.w. This patient had a progressive hearing loss for nine years with characteristic clinical and audiological findings for otosclerosis.

On May 12, 1956, an operation was performed to achieve mobilization of the stapes. It was firmly fixed, and forces applied to the capitulum and footplate resulted in crural fracture. The hammer was placed on the footplate, two blows were delivered and the second blow resulted in a fracture of the footplate. Surgical audiometry showed improved thresholds for the low frequencies, but greater loss for the high frequencies.

SURGICAL AUDIOMETRY.

	500	1000	2000	4000	6000
Immediately before hammer	55	65	55	50	45
Immediately after hammer	30	50	65	75	65

Subsequent tests have shown this loss to involve both bone and air conduction thresholds and presumably is due to inner ear damage. Several tests during the subsequent seven months have shown no recovery, so that injury must be considered permanent (see Fig. 3-B).

C. ROUND WINDOW OTOSCLEROSIS.

The study of temporal bones of patients having either histologic or clinical otosclerosis suggests that 30 to 40 per cent of all patients with otosclerosis have involvement of bone in the region of the round window niche.[2-6] Both histological and clinical observations indicate that complete closure of the round window niche is rare. Animal experimentation shows that auditory thresholds are not affected by partial closure of the round window; in fact, a pin-point opening appears to be sufficient to preserve normal cochlear hydrodynamics.

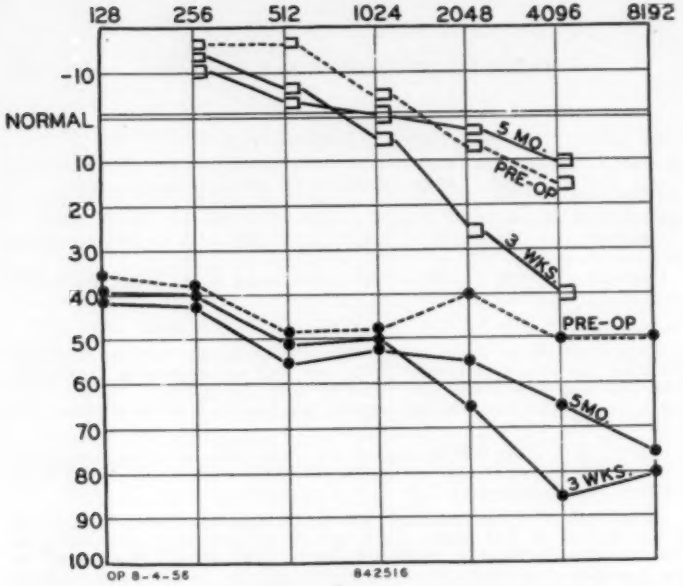
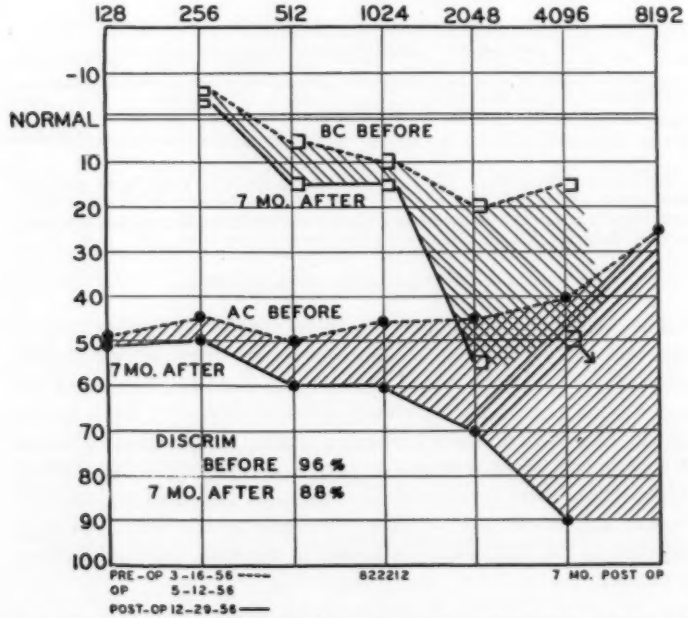


Fig. 3-A. Case 2. Audiograms showing temporary cochlear injury as a result of using a hammer on a ankylosed stapedial footplate.



B. Case 3. Audiograms showing permanent cochlear injury from use of a hammer on the stapedial footplate.

Lindsay and Hemenway have reported an interesting human case in which thresholds were unchanged, although four-fifths of the normal area of the round window was blocked by otosclerotic bone.[7]

Some otologists believe that otosclerotic closure of the round window is responsible for certain failures following fenestration surgery. This might account for failure to improve hearing, although good bone conduction thresholds were retained, and an active fistula response was present.

The case history of a patient with complete obstruction of the round window as determined by direct observation at the time of surgery follows:

Case 4. A.B.—No. 710407—age 34, f.w. The patient had a hearing loss for ten years which became slowly but progressively worse. For four years she had occasionally short-lasting attacks of unsteadiness which were not severe enough to be incapacitating. Her father, paternal uncle and brother had partial hearing losses which began in middle age. The patient had fractures of the right femur at the ages of 2, 4, 13 and 28, ribs at age 24 and nose at age 31. She has two children, both of whom have had multiple fractures.

General physical examination was not remarkable except for bilateral blue sclerae. The tympanic membranes appeared normal. Audiometric examination showed a bilateral conductive type hearing loss and a moderate elevation of bone conduction thresholds for high frequencies.

On June 6, 1956, at the time of operation on the left ear the stapes and long process of the incus were found to be freely mobile; however, the round window niche was filled with a mass of bone (see Fig. 4-A). The mucosa was elevated from this otosclerotic bone and from the adjacent promontory, but no opening could be found. The otosclerotic bone was very firm, and would not yield to gentle curretting. No further attempts were made to open the round window niche. Surgical audiometry showed a decrease in thresholds.

SURGICAL AUDIOMETRY.

	500	1000	2000	4000	6000
Before exploration	40	45	60	60	65
After exploration	65	65	65	60	75

Subsequent tests have shown consistently worse thresholds (see Fig. 4-B). We cannot explain this as we would have predicted no change in thresholds.

The question might be asked, "In such cases can the round window niche be re-opened without injury to the cochlea?" If we assume that the otosclerotic bone forms a thick cur-

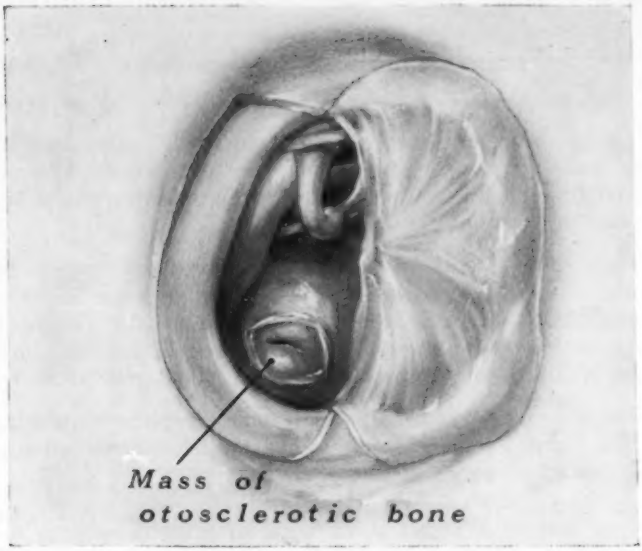
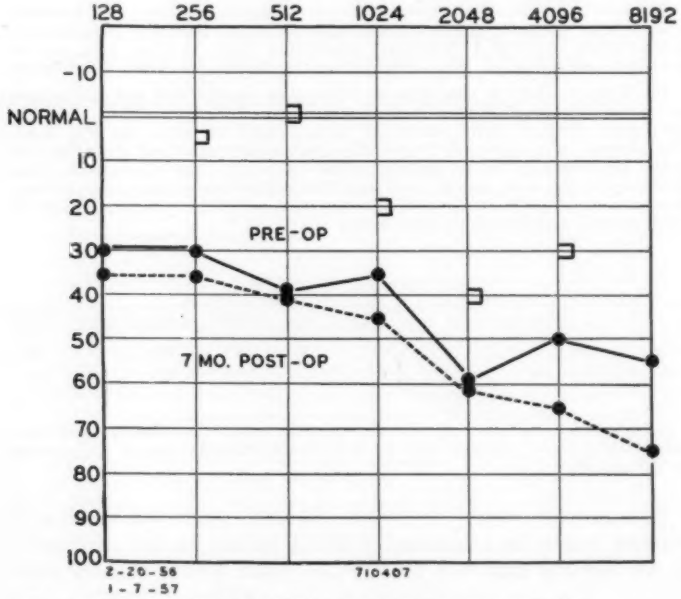


Fig. 4-A. Case 4. Upon operation a mass of otosclerotic bone was seen filling the round window niche as shown in this drawing. The mucosa was reflected from the otosclerotic bone and adjacent promontory, but no opening could be found into the round window niche.



B. Audiograms showing slightly greater loss after operation.

tain overhanging the round window membrane, then part of it could be removed safely. On the other hand, if the new bone has grown out in the region of attachment of the round window membrane so as to encroach upon it and replace it, then removal would result in opening the scala tympani. A small tear in the round window membrane probably would not damage the inner ear, if gel-foam were placed over it. For example, in experiments on cats we have created small tears in the round window membranes and covered the openings with gel-foam and observed healing without injury to the inner ear.

In the case described by Lindsay and Hemenway the round window membrane was actually replaced by the encroaching mass of otosclerotic bone. Should such a process continue to complete closure, there probably would be no safe way of re-establishing the opening.

When performing stapes mobilization some otologists apparently make a routine practice of trimming or removing visible otosclerotic growth from the margin of the round window. This maneuver would seem to be of doubtful value for two reasons; first, only a pin-point opening is necessary to preserve the normal hydrodynamics of the cochlea and, second, complete closure of the round window is rare. Likewise, connective tissue bands stretching across the round window niche probably have no effect on hearing unless they create a dense, complete blockage of the niche.

D. ELONGATION OF THE PYRAMIDAL EMINENCE.

It is possible for the stapes to be ankylosed by a bridge of bone extending from the pyramidal eminence to the neck of the stapes. The case history of a patient with this disorder follows:

Case 5. B.T.—No. 490913—age 55, f.w. This woman had a progressive loss of hearing for 12 years. She had worn an air conduction hearing aid in the left ear for five years.

The skin of the left ear canal was thickened as a result of irritation from the ear mold. Audiometric study showed a bilateral conductive type deafness which was worse in the left ear. Bone conduction thresholds were normal.

When the operation was performed on June 30, 1956, the stapes was

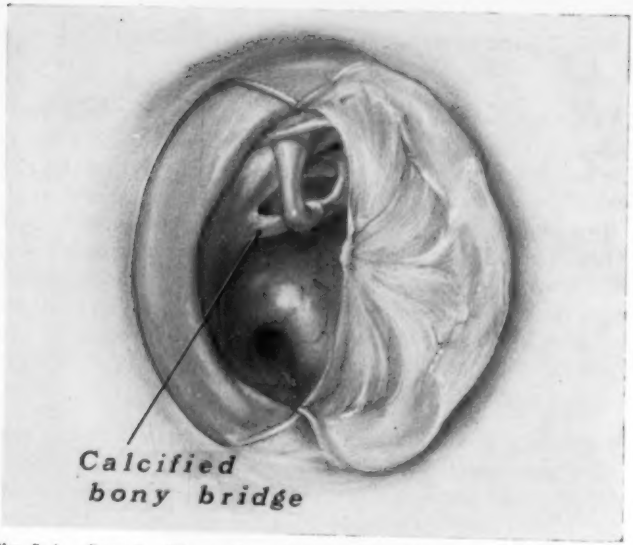
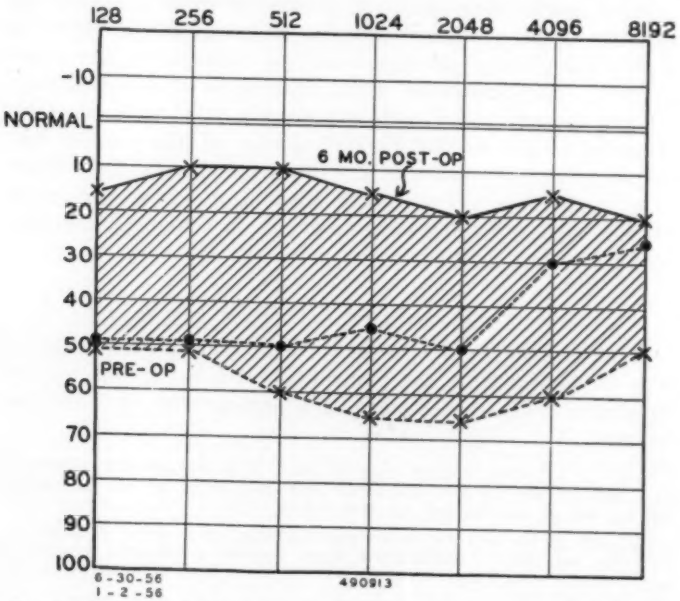


Fig. 5-A. Case 5. Elongation of the pyramidal eminence had ankylosed the stapes.



B. Auditory thresholds were greatly improved after removal of the bridge of bone.

found to be ankylosed by a bridge of bone extending from the pyramidal eminence to the neck of the stapes so as to immobilize it. (see Fig. 5-A). When this bridge of bone was removed with a small curette the stapes became mobile, and auditory thresholds improved. The footplate did not appear to be ankylosed.

SURGICAL AUDIOMETRY.

	500	1000	2000	4000	6000
Before	55	60	70	60	60
After	30	35	50	55	60

The improved thresholds have been maintained to date (see Fig. 5-B).

E. ONE-STAGE BILATERAL MASTOIDECTOMY.

During the past two years we have performed mastoid surgery for chronic suppuration on 82 patients. In this group were 11 patients (or 13 per cent) with bilateral suppuration requiring bilateral mastoid surgery. The ages of these patients varied from 6 years to 47 years, and the duration of suppuration varied from 3 years to 42 years. The mastoid cavities were skin grafted in nine of the 11 patients.

We wish to direct attention to the question of whether surgery should consist of a one-stage operation, or whether surgery on the second ear should follow the first by an interval of days or weeks. Our approach to the problem has been to handle all bilateral cases at a one-stage operation.

Of the 11 patients, six had bilateral modified radical mastoidectomy, four had a modified radical mastoidectomy on one ear and a radical mastoidectomy on the other, and one had bilateral radical mastoidectomies.** When surgery on one ear had been completed the patients were allowed to enter a light stage of anesthesia so that facial movements could be examined. The total operating time varied from two hours, 45 minutes, to five hours (average four hours, seven minutes). The hospital stay varied from seven days to ten days (average eight-and-one-half days).

**By "modified radical mastoidectomy" we mean all procedures designed to utilize part or all of the conduction system, or to accomplish a condition favoring sound pressure differential between the two windows. "Radical mastoidectomy" includes all other cases.

In weighing the relative merits of the one-stage and two-stage operations, several factors must be considered:

1. Anesthetic risk: We have been unable to acquire data on the relative risk, for example, of a four-hour operation to that of two two-hour operations. The operative risk is so small and the difference is probably so slight that this factor deserves little consideration.

2. Convenience for the patient: If promised equal end-results all patients prefer a one-stage operation. Pre-operative anxiety and post-operative discomfort, though mild, need be experienced only once. There is no significant increase in post-operative discomfort when both ears are operated upon. The loss of time from work is half as great, and the total number of post-operative visits are fewer when a one-stage operation is done.

3. Surgeon's preference: The bilateral mastoidectomy might seem excessively long and tiring for some surgeons.

4. End-Results: An equally good end-result should be acquired whether the procedure is one-stage or two-stage. We can see no advantage to waiting for the end-result of one operation before performing the second.

5. Tympanoplasty: Henceforth, most ears with chronic suppuration will receive some form of tympanoplasty in an attempt to restore hearing. These procedures will prolong the operating time. We believe that if the operating time on the first ear is longer than three hours it would be better to perform the second ear at a second stage. On the other hand, because of the rather prolonged after-care required for some patients undergoing tympanoplasty, there would be a definite advantage to treating both ears at once.

SUMMARY.

Several interesting middle ear problems have been encountered recently:

1. A surgically dislocated incus of long standing in a boy of 16 was replaced with temporary excellent improvement in hearing. During the subsequent nine months, however, the

auditory acuity has gradually diminished, which suggests a progressive subluxation of the incus.

2. The injudicious use of hammer blows to a rigid stapedial footplate can create an acoustic impulse of sufficient intensity to injure the organ of Corti in the basal turn.

3. A hard-of-hearing patient was found to have complete blockage of the round window by a mass of otosclerotic bone.

4. The stapes can be ankylosed by elongation of the pyramidal eminence so as to create a bony bridge from the posterior wall of the tympanum to the neck of the stapes. Removal of this bridge results in hearing improvement.

5. For chronic bilateral middle ear and mastoid suppuration requiring surgery a one-stage bilateral operation is preferable to two separate operations, providing the total operating time is not greater than four or five hours.

We are grateful to Curl Tutag, Ruth Shaw and Rosemary Doran for assistance with the manuscript, and to Elton Hoff for the drawings.

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SPEECH INTELLIGIBILITY IN RELATION TO THE SPEED OF THE MESSAGE.*

C. CALEARO, M.D.,
and
A. LAZZARONI, M.D.
Milan, Italy.

INTRODUCTION.

The amount of information, (I) contained in a speech wave may be expressed by the equation $I = 2\ tw \log \frac{S}{N} + N$, where t is the time in seconds, w the width of the speech frequencies band, S the amplitude of the signal, N the value of a just noticeable difference in intensity (Miller).

It is conceivable that, by varying one of the factors in the aforesaid equation, the others should vary correspondingly in order to keep the value of the equation unchanged. If, for instance, the time during which the signal is present is reduced, some increase in intensity presumably will be necessary in order that the same amount of information may be contained in a given speech wave.

Under ordinary conditions, the redundancy of the elements of information contained in an ordinary speech wave is such as to insure a perfect intelligibility of a message, even though only one of the factors of the equation varies. In case of increase of the speed of delivery of speech, small increases will be more than compensated by the principle of redundancy, but further increases must probably be compensated by a simultaneous increase in intensity.

It has been our aim to arrive at a clear definition of the relationship between speed of delivery and intensity in speech discrimination both under normal and pathological conditions.

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*From the Department of Otolaryngology of the University of Milan, Italy (Director: Prof. L. Pietrantoni).

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MATERIAL AND TECHNIQUE OF THE TEST.

In our experiments, we have used lists of short significant sentences, which have been recorded at three different speed rates. The first one (140 words per minute) may be considered as the normal rate of speaking for the Italian language. The other two recordings were made at a rate of 250 and 350 words per minute.

These three different rates of acceleration have been obtained in three ways:

1. By using a speaker having exceptional possibilities of accelerating his speaking rate.

2. By transferring the primitive recording on a magnetic tape which could be rotated at different speeds.

3. By employing a special apparatus which allows a direct acceleration of the message without any alteration of frequency.

A comparison between the results of these three methods has shown that there are no significant differences between them, so we have used them promiscuously in our tests.

The message was delivered through headphones to the subject, isolated in a sound-proof room, and he was instructed to repeat the sentences as he heard them.

DISCUSSION OF RESULTS.

The curves of Fig. 1 show the average articulation score obtained in five subjects with normal hearing for three different syllabic-rates.

The threshold shift, that is to say the displacement of the curve from normal values owing to the acceleration of speech, reaches 5-10 db at a rate of 250 words per minute and 10-15 db at the rate of 350 words per minute; however, the shape of the curves remains unchanged: the three curves obtained at progressively increasing syllabic rates run more or less parallel to each other; therefore, in a normal subject, the impairment of discrimination due

to an increase of the syllabic rate per minute is almost completely neutralized by a simultaneous slight increase of intensity. This experimental evidence supports the views expressed in the introduction note.

We have extended these research methods to a group of subjects who presumably suffered from an impairment of the activity of the central mechanisms of association and correlation with a possible increase of the latency time of the central interneuronic reactions on which the identification mechanism depends. In these cases, it could be supposed

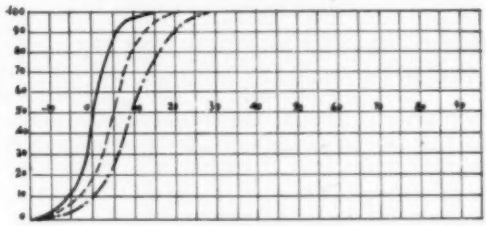

Fig. 1. Articulation scores for continuous speech, switched periodically from one ear to the other. For each ear the proportion of the period occupied by speech is 50 per cent, the remainder being silent. Results for three rates of speaking are given. (after Colin Cherry and Taylor)

that the central variation of the time factor might be sufficiently neutralized by the said redundancy of the elements of information contained in the message only when the speed of delivery of speech remains within normal limits. On the contrary, it could be inferred that, by superimposing the central delay of identification to a peripheral acceleration of the message, the combination of these two phenomena would result in speech discrimination in a marked impairment of discrimination. Furthermore, the normal relationships between speed of delivery and intensity would be substantially altered.

Fournier (1954) has already stressed the importance of the time factor in discrimination. In his opinion the time required for the complicated process of central elaboration of the message may be presumed to amount to one-tenth of a second, considering that the phonemes are delivered in

normal speech at a rate of approximately ten per second, and that an increase of this rate ensues in a greater difficulty in following a speech or a conversation. According to Fournier, a lengthening of the time required by the cortex for the identification of the message might account for some aspects of the troubles of speech perception in aged persons.

In the aged, where presbycusis is most probably due to the involution of many central as well as pheripheral structures, it may be inferred that there exists a delay at the level of the various synapses of the auditory pathways and of the cortex.

Bordley and Haskins (1955) have shown that when words are presented to old people at a high average syllabic rate there results an increased difficulty of intelligibility.

Finzi (1955) has shown in a series of systematic tests with accelerated speech, that in the aged the intelligibility threshold may be reached only rarely, and that the threshold shift is much increased, if compared to the one of normal subjects. He has observed that, in the majority of cases (15 out of 25), no intensity level allows a 100 per cent intelligibility at a rate of 350 words per minute.

It has been our intention to find out the precise discrimination scores as a function of intensity for messages recorded at different syllabic rates.

For this purpose, we have submitted to our test of accelerated speech, a group of six aged subjects ranging from 70 to 85 years, in good general condition and normal from the psychological point of view. They all came from an old pensioners' home. Their pure-tone audiogram was within the limits of "physiological" presbycusis, their speech threshold corresponding as closely as possible to the pure-tone curve, and their articulation curve was of normal shape and steepness.

The curves of Fig. 2 are quite typical. They represent in the same subject the three articulation curves for a message delivered at the three different speeds. The thresh-

old shift appears to be much increased (30 db) even for the intermediate speed, while by testing the patient with the speech at the highest speed it was not possible to attain even the threshold of perception (50 per cent discrimination) at any intensity level.

Figs. 3-4-5 show a comparison between the articulation curves for normal, medium and high speed rates in the six cases studied, and in the normal subjects.

From the evidence and consistency of our results we have not thought it necessary to perform further tests on a greater number of cases.

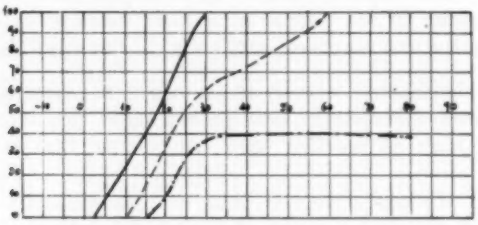

Fig. 2. Three articulation curves obtained in the same aged subject for a message delivered at three different speeds.

The test with accelerated speech has been employed in another research on central deafness which we are performing in collaboration with Bocca.

Assuming that a central lesion may lead to a delay of the normal synaptic time, one could foresee an increased difficulty in understanding a message read at a high syllabic rate whenever a tumor, whether or not accompanied by increased intracranial pressure, affects the auditory cortex.

Our constantly positive findings in a group of ascertained intrinsic lesions of the temporal lobe have shown that this type of distortion may be considered as a valuable test for diagnosis of an impairment of the auditory area and of the side of the lesion. As a matter of fact, the articulation curve is clearly worse when the accelerated message is sent

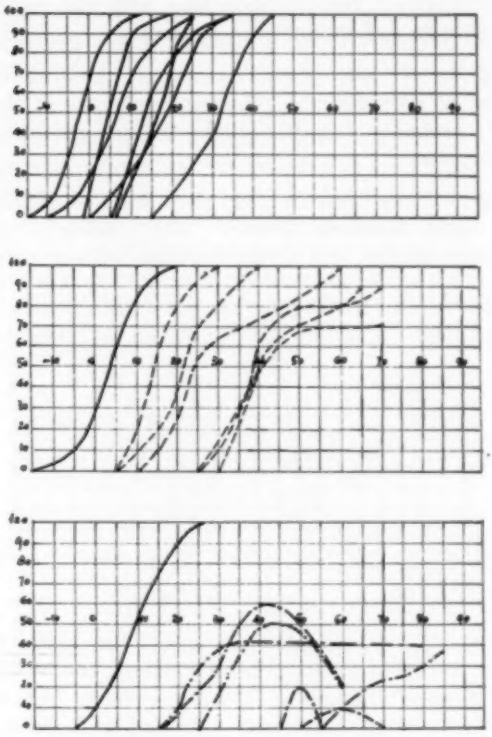

Fig. 3, 4, 5. Articulation curves for normal, medium and high speed rates in six subjects aged over 70, compared to the one of the young subjects (first curve).

to the ear contralateral to the lesion. This finding is in accordance with the results of other special audiological tests, which had already been performed in order to study the disturbances of elaborate hearing at a psychological level in cases of pathology of the temporal cortex.

The cases with severe intracranial pressure showed an enormous and sometimes infinite threshold shift. This phenomenon was present on both sides when the tumor did

not directly affect the temporal lobe and was more marked in the opposite ear in intrinsic tumors.

In a group of cases of extra-temporal cerebral tumors we could observe only a slight symmetric increase of the threshold shift, probably due to a slight diffuse suffering of the central organs.

The curves reported in Fig. 6 show the findings in a case of right temporal tumor. The intelligibility of the accelerated message sent to the homolateral ear may be con-

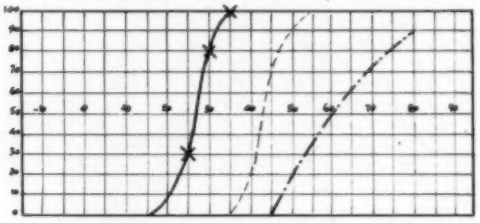

Fig. 6. Articulation curves for the left ear at different speed rates in a case of right temporal tumor.

sidered as normal. On the contrary, the threshold shift for the message at intermediate speed in the contralateral ear was slightly increased (20 db). When the test was performed, by using a message recorded at highest syllabic rate, the threshold shift, if compared to normal, was quite remarkable, and it was not possible to reach a 100 per cent intelligibility at any intensity level.

Fig. 7 shows the three articulation curves for three different syllabic rates in a case of tumor of the left temporal lobe accompanied by severe increase of intracranial pressure. Articulation is bilaterally poor even at intermediate speed.

It is interesting to note that a comparison of these findings with those obtained in aged persons shows that in the latter group the threshold shift is constantly higher than in those suffering from cerebral tumors with exception of

those complicated by severe intracranial pressure, where the curves are very much similar to those observed in the old age group.

From these observations, we may deduce that an impairment of intelligibility for accelerated speech may, on one hand, stand for a cortical suffering and even allow a localization of the process in one hemisphere, but also that it depends, to a high degree on a lengthening of the synapsis time along the entire central auditory pathways. A proof of this diffused alteration is offered not only by the said difference between findings in temporal tumors and in the aged, but also, by the similarity of data observed in old

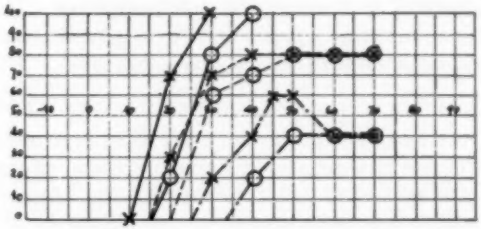

Fig. 7. Articulation curves for three different syllabic rates in a case of tumor of the left temporal lobe accompanied by severe increase of intracranial pressure.

persons and of those with severe intracranial pressure, which is bound to compromise the function of auditory path well below the cortical level.

At the close of this discussion, we venture the conclusion that the study of the articulation scores as a function of the speed of delivery of speech may be useful for an investigation of the superior auditory functions, showing trouble in the elaboration and identification of the message which may escape attention with the routine methods of examination. An increase in the speed delivery of speech puts a strain on the centers, and the effort of discrimination is possible only when the superior hearing mechanisms are unimpaired; therefore, we feel that the accelerated speech

test can be adopted in audiological practice and may throw further light on hitherto unknown aspects of hearing at a superior level.

SUMMARY.

The authors have studied the intelligibility of speech as a function of the speed of delivery.

Experiments on normal subjects have shown that the redundancy of the superior structures and of the information contained in a speech message allow a nearly complete neutralization of the negative effect due to the increased speed of delivery when such increase is contained within modest limits. For further increases of speed, a rise of intensity is needed to insure good discrimination. The normal relationships between speed and intensity in speech discrimination have been carefully investigated.

The research has been extended to subjects showing alterations of the auditory path and centers with a normal hearing function as examined with routine tests (aged persons and patients suffering from tumors of the temporal lobe). In these cases, the impairment of the function of superior association and correlation mechanisms, consisting in a lengthening of the reaction time of auditory path and centers, leads to an impairment of intelligibility of accelerated speech. The articulation thresholds and curves for variously accelerated messages are then considerably different from normal.

In cases of pathology of the auditory cortex, the test with accelerated speech may further allow a specific localization of the side of the lesion, as due to the prevalently contralateral projection of each cochlea, the hearing defect is greater in the ear opposite to the lesion.

Acknowledgments.

We are much indebted to Prof. E. Bocca for his help and advice, to the Italian Broadcasting Company (RAI) and to the Director of the Department of Musical Phonology of RAI, maestro L. Berio, for active collaboration in special recordings needed for this research.

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SOUTH CAROLINA SOCIETY OF O. & O.-L., AND NORTH CAROLINA E. E. N. & T. SOCIETY JOINT MEETING.

The joint annual meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose, and Throat Society will be held in Hendersonville, N. C. The dates are September 15, 16, 17, 18, 1957. Headquarters will be the Skyland Hotel. The following guests ophthalmologists will be on the program:

Dr. E. A. Naumenee of Baltimore, Md.; Dr. P. G. Leinfelder of Iowa City, Iowa; and Dr. Alston Callahan of Birmingham, Ala. Likewise the following otolaryngologists will be on the program:

Dr. Francis LeJeune of New Orleans, La., and Dr. J. W. McLaurin of Baton Rouge, La. The third otolaryngologist will be announced shortly.

Hendersonville, N. C., is located in the Blue Ridge Mountains, and is a particularly lovely spot at this season of the year. A large attendance is anticipated. For further information write Dr. Roderick MacDonald, Secy.-Treas., 330 East Main St., Rock Hill, S. C.

THE EVOLUTION OF A STAPES MOBILIZATION TECHNIQUE.*

EUGENE L. DERLACKI, M.D.,
GEORGE E. SHAMBAUGH, JR., M.D.,
and
WILEY H. HARRISON, M.D.,
Chicago, Ill.

This is a preliminary report of the combined experiences of three otologists in evolving a technique for stapes mobilization surgery as an adjunct to fenestration surgery for the treatment of clinical otosclerosis. It includes 440 operations during the 25 months between September, 1954, and November, 1956.

In the past year reports by Belluci,[1] Goodhill,[2] the Meurmans,[3] and Kos[4] and Scheer[5] have added to the articles previously contributed by Rosen after his original report[6] in 1952, of accidental mobilization of the stapes footplate while testing for stapes ankylosis in otosclerosis. Rosen's purposeful mobilization of the stapes, as reported in 1953, met with considerable initial skepticism which is gradually being dissipated in the light of later reports by these authors and others. An historical review of the 19th Century successes and failures in surgical efforts to mobilize the stapes, followed by oblivion for 50 years, has been adequately documented during the current Renaissance and does not require recapitulation.

Our own skeptical interest in Rosen's work changed to a more open-minded and active state after two of us, prompted by Dr. Philip MacDonald,[8] observed Rosen in several operations in September, 1954, and again in December, 1954, with the opportunity to examine and test several of his postoperative cases.

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*Read before the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Cleveland O., January 10, 1957.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 17, 1957.

After a discouraging first six months, during which we attempted to follow Rosen's originally described technique, we have gradually evolved a technique in which we have greater confidence and with which we are obtaining an appreciably higher percentage of initial successes. Fully aware of the preliminary and tentative nature of the results thus far reported, including our own results reported here, we are presenting our early experiences as a possible help to others who are engaged in this challenging work.

EVOLUTION OF SURGICAL TECHNIQUE.

Our first operations followed as closely as possible the technique originally described by Rosen.[7] This was soon found to be frequently unsuccessful in our hands, the chief disadvantages being the relatively blind tactile approach and frequent fracture of the crura using Rosen's mobilizer and maneuver. The personal observations, by two of us, of Kos using a fine-tipped slightly angled microscopic hook, commonly used to clean the fenestra, as a palpator and stapes mobilizer, led to our first important modification. We changed the standard 15° (Shambaugh) microscopic hook in three ways. The angle of the pointed tip was further straightened to 10°, the pointed tip was sharpened, and the shaft was lengthened for use through our ear speculum under the long working distance of the new Zeiss binocular operating ear microscope. The spring in the shaft was kept the same as in the fenestration hook by slight thickening of the base near the handle.

Our long experience in fenestration surgery under the operating microscope[9] led us to utilize the added magnification of the new Zeiss instrument with its illumination directly through the objectives as an indispensable routine in our stapes mobilization operations. With 16-X magnification and removal of the posterior-superior bony meatal rim we found that a beautifully detailed view of the footplate could be obtained from above, with clear visualization of footplate mobility during and following successful manipulation.

The next step in the development of our technique was the application of mechanical principles to the direction of

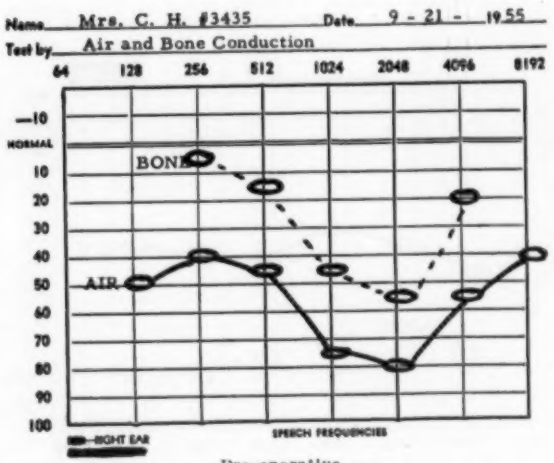
the mobilizing force, and the construction of a new blunt ended mobilizer (E.L.D.).

In presenting our current technique we acknowledge the many contributions of others, especially of Rosen, and of the stimulus and helpful ideas gained by two of us from observing operations by Kos, Goodhill, and House.

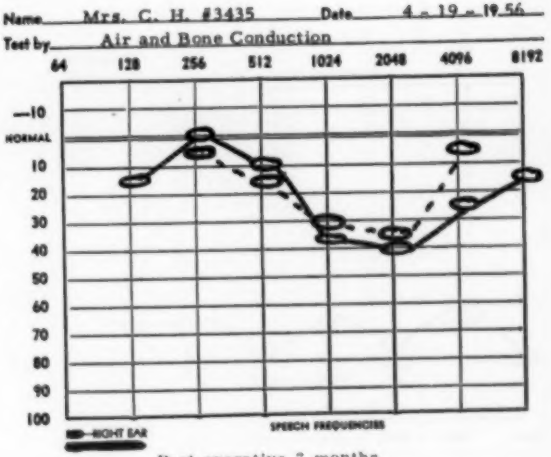
DISCUSSION WITH THE PATIENT.

Our changing attitude toward mobilization is evidenced by our preoperative discussion of surgery with our patients with otosclerosis. Two years ago we discouraged patients who expressed interest in stapes mobilization. If the patient wished to try this experimental procedure we would do it, but did not advise it. Our attitude was reflected in our first 100 mobilization operations, in that cases of poor suitability for fenestration ("C" classification) outnumbered the total of ideal and good suitability cases ("A" and "B" classification).

Our present discussion offers the patient a 40 to 50 per cent chance of initial success, with mobilization recommended as a preliminary procedure to fenestration for the majority of patients of "A" and "B" suitability, as well as for cases of "C" suitability. Only when a patient appears temperamentally unsuitable for a two-stage operation do we advise fenestration first. We bring out the fact that the success of mobilization depends as much upon the extent and pattern of the otosclerotic involvement of the footplate as upon the surgical technique, so that the outcome of this procedure in a particular patient cannot be predicted until the stapes has been exposed and examined. For this reason we refer to stapes mobilization as a preliminary exploratory operation, to be followed by fenestration six months later when the extent of otosclerotic involvement prevents mobilization or causes refixation. *In no way should stapes mobilization be presented as a substitute or alternative to fenestration.* In our discussion we also emphasize that stapes mobilization is a relatively new procedure, with little or nothing known as yet concerning the ultimate duration of the hearing improvements; and, as in our discussion of fenestration, we include

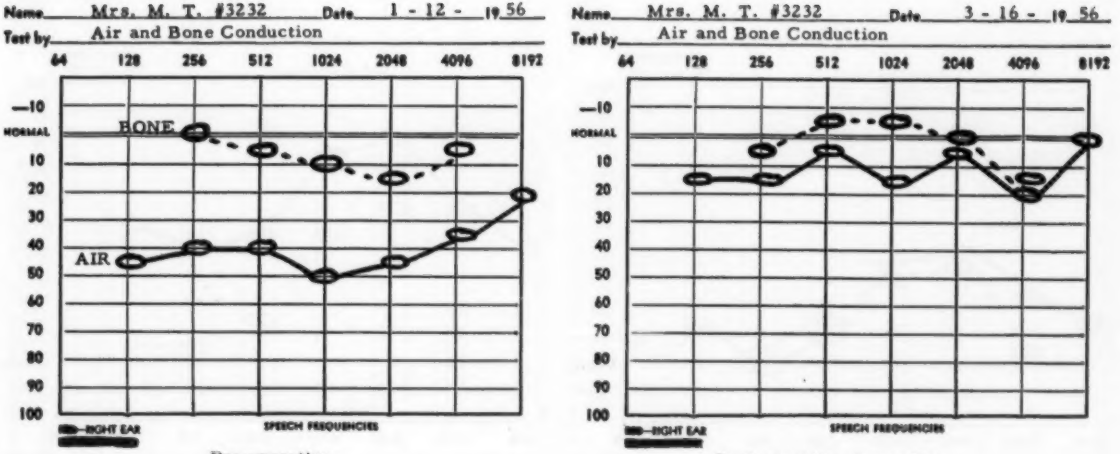


Pre-operative.



Post-operative 7 months.

Fig. 1. Eradication of air-bone gap by stapes mobilization in "C" case for fenestration. (Note improvement in bone conduction due to reversal of Carhart notch).



Pre-operative.

Post-operative 2 months.

Fig. 2. Result from mobilization of "A" Case better than predicted from fenestration. Note perfect pre-operative Carhart notch of stapes ankylosis reversed by liberating the stapes.

the possibility of a further loss in the operated ear when the operation is unsuccessful.

PRESENT STATUS OF SURGICAL TECHNIQUE.

Indications. At the present time there are no absolute criteria for prediction of result and case selection for stapes mobilization as there are for fenestration. The residual con-

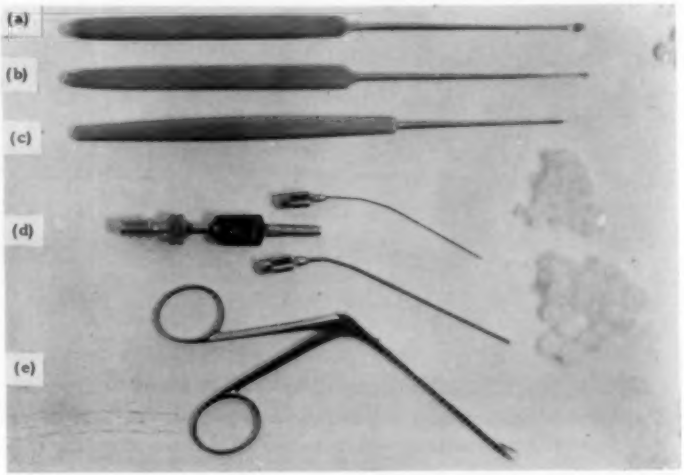


Fig. 3. Instruments for stapes mobilization modified by the authors: (a) "Duck-bill" knife and elevator (Kos); (b) angled knife and elevator (Kos); (c) fine tipped "monkey" periosteal elevator (Shambaugh); (d) fine suction tips and adaptor; (e) "alligator" scissors (House).

ductive loss due to interruption of the ossicular chain following fenestration that averages 25 db., or 20 db. when the tympanic membrane is blocked,[10] permits a reasonably accurate and reliable prediction of result from fenestration in each patient with otosclerosis.[11] Theoretically, fully successful stapes mobilization should not leave a residue of unrestored conductive loss, the cochlear function being the ceiling to the hearing result in a given case.[12] Actually, contrary to the impression conveyed in some of the published articles, this ceiling is only occasionally attained (see Figs. 1, 2, 10), most

cases of "successful" stapes mobilization having a variable residue of conductive loss, probably because the mobility of the ossicular chain has not been fully restored.

In any eventual system for the selection of ideal, suitable, and borderline cases for stapes mobilization it seems reasonable to include both the level of cochlear reserve and the degree of stapes fixation as measured by the air-bone gap and tuning forks.

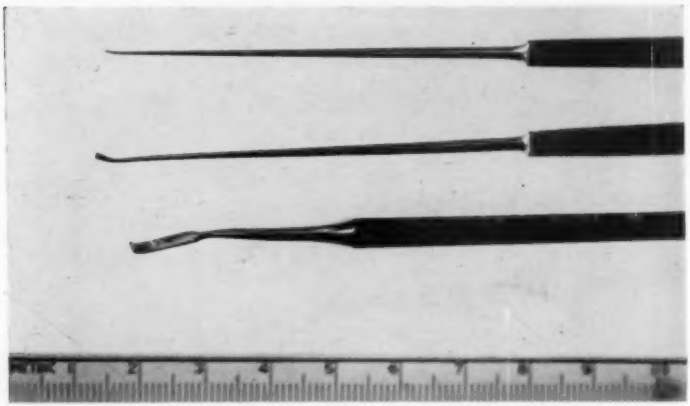


Fig. 4. Instruments for stapes mobilization introducted by the authors: Top—modified straight 10° Shambaugh microscopic hook mobilizer; middle —flattened end Derlacki mobilizer; lower—dental scaler used as a bone curette for exposing the footplate to view.

Stapes mobilization can be used for patients with partial fixation or an air-bone gap of only 20 to 25 db., unsuitable for fenestration because of the expected residue of unrestored conductive loss after the latter procedure. Stapes mobilization can also be tried in cases with advanced nerve degeneration combined with a good air-bone gap, where complete eradication of the air-bone gap can be hoped for, with a better hearing result than fenestration could offer (see Fig. 1).

All three of us would choose stapes mobilization on ourselves as a preliminary procedure to fenestration, were we to have otosclerosis of "A" or "B" suitability. We would

choose it because of the definite (though not great) possibility of a higher level of hearing than could be obtained by fenestration (see Fig. 2), because of the minimum disability and because of the absence of a postoperative cavity requiring periodic lifetime care.

Preoperative Preparation. The patient enters the hospital the evening before surgery, having been provided with a printed sheet explaining the preliminary exploratory opera-

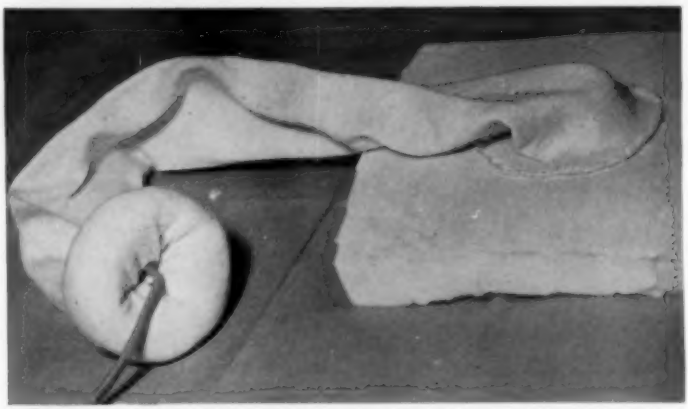


Fig. 5. Sterilized sponge rubber ring and cloth sleeve (Rytzner) containing audiometer ear piece and cord.

tion, the approximate chances of success or failure in obtaining a useful hearing result by mobilization of the stapes, and if the case is suitable for fenestration, the fact that fenestration will follow six months later if mobilization is unsuccessful.

Preoperative sedation with nembutal 0.1 gm. (gr. 1½) one hour before operation allays anxiety but does not prevent good cooperation in audiometric testing.

We believe that too little stress has been accorded preoperative invasion of a clean middle ear *and inner ear* when a successful mobilization of the footplate is accompanied by escape of perilymph. We feel that rigid aseptic technique

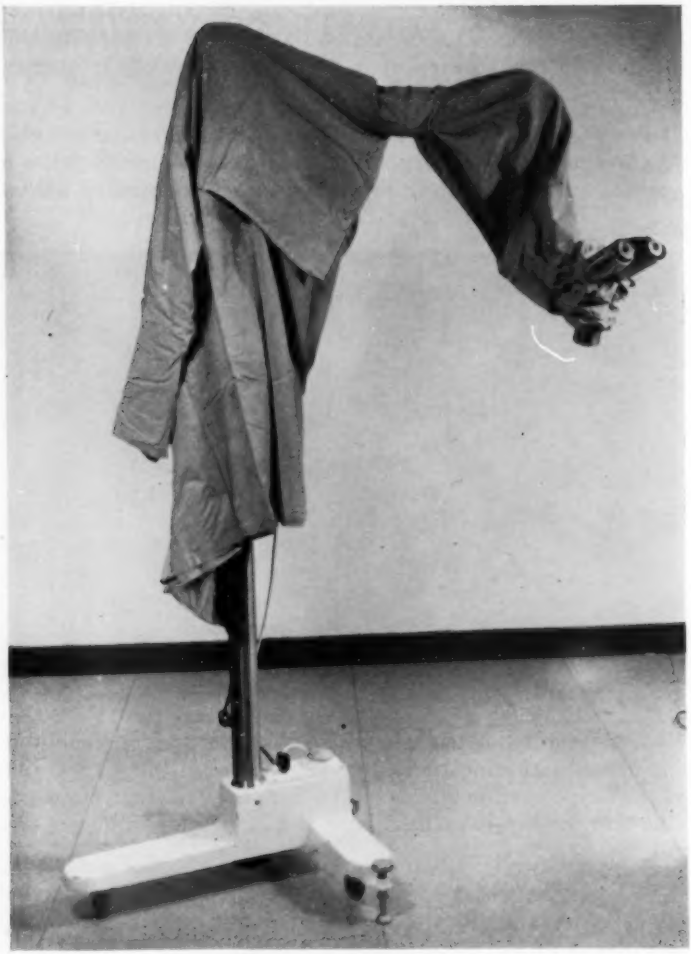


Fig. 6. Zeiss operating microscope protected by sterile drapes and sterile metal hoods over the oculars and eye pieces.

is more important than prophylactic chemo- or antibiotic therapy in preventing postoperative external otitis, otitis media, or the much more serious suppurative labyrinthitis. We suggest that postoperative complications from infection contributed toward the abandonment of stapes mobilization attempts at the end of the last century.

Before the patient is brought to surgery, the hair from in front of the ear to the upper edge of the auricle is shaved. Prior to *prepping* the ear in the operating room two inch-

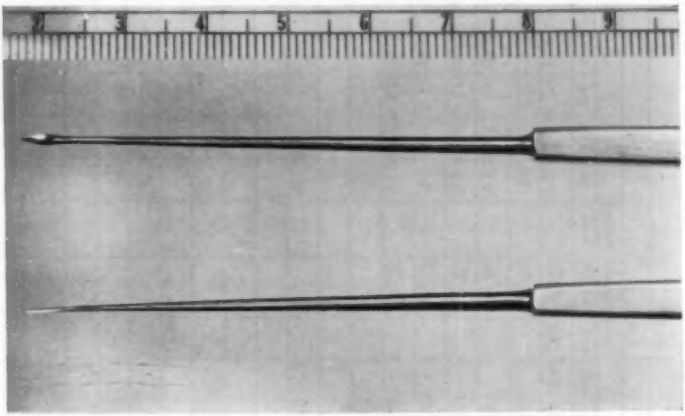


Fig. 7. Instruments for stapes mobilization introduced by the authors: Upper—angled capsule knife (Derlacki); lower—fine footplate chisel (Derlacki).

wide strips of Scotch tape are placed above and behind the ear to keep hair out of the field. The external auditory meatus is filled with colorless tincture of Merthiolate 1-1,000 and the auricle, surrounding skin and Scotch tape, are scrubbed three times with sterile gauze sponges and soap containing hexachlorophene, always working from the meatus toward the periphery. Three applications of 70 per cent alcohol, followed by colorless tincture of Merthiolate to all exposed surfaces and sterile drapes are applied. Throughout the procedure, all of the operating room personnel adhere to a rigid sterile technique exactly as in fenestration surgery.[9]

Pt's Name MR. R. H. #3598 | Rt. Ear

Surgeon DR. D. | Left Ear

Date	RIGHT EAR 64	128	256	512	1024	2048	4096	8192	Average for 512, 1024, 2048	Post-op change in db.	LEFT EAR 64	128	256	512	1024	2048	4096	8192	Average for 512, 1024, 2048	Post-op change in db.
		60	50	55	55	40	55	20	50		PRE-OP OFFICE									
A			65	60	60	50			57		(M. T. ELEVATED AND REPLACED)									
B			55	50	45	40			45		STAPES MOBIL. (M. T. IN PLACE)									
C			40	30	35	40			35		STAPES MOBIL. (M. T. IN PLACE)									
D			70	65	65	70			67		STAPES MOBIL. (M. T. IN PLACE)									
E			35	25	35	35			32		STAPES MOBIL. (M. T. IN PLACE)									
			10	5	15	10	-5				BONE									

Fig. 8-A. Audiometric tests during surgery with various positions of the mobilized stapes and dislocated incudo-stapedial joint.

Infiltration and Incision. The anesthetic solution used is six parts of 2 per cent xylocaine to one part of adrenalin 1-1,000. Local infiltration is begun in the posterior meatal wall just lateral to the osseous meatus followed by injection of the inferior, anterior, and superior meatal walls, an approximate total of 1.0 cc. of solution being used. Infiltration of the posterior wall especially is done slowly to prevent bleb formation.

The entire surgical procedure is performed through an ear speculum, as described by Rosen.[7] We have found that an endaural incision to enlarge the field offers no particular advantage.

The skin of the posterior osseous meatal wall is incised from approximately 12 o'clock to 6 o'clock, beginning close to the annulus and swinging lateralward a good 5 mm. from the annulus in most of its U-shaped course. The incision and initial elevation are carried out with the same "duck-bill" instrument (see Fig. 3) which we have modified from Kos, down to the sulcus tympanicus where the fibrous annular ligament of the tympanic membrane lies in the bony groove. A dry field is secured by placing tiny cotton balls moistened in the xylocaine-adrenalin solution between the elevated skin and the bone, and applying suction. Any remaining bleeding points are controlled by electro-cautery applied to the fine suction tip (see Fig. 3). The annular ligament is then elevated from the sulcus with the angled elevator (see Fig. 3), again modified from Kos, gently pushing this instrument directly inward at repeated points along the sulcus. Finally, the delicate middle ear mucosa is separated, and the tympanic membrane is folded forward upon itself, using the tiny periosteal elevator designed by one of us (G.E.S) for elevating the tympanomeatal flap in fenestration of the monkey ear (see Fig. 3). The posterior portion of the middle ear is now exposed from about 11 o'clock in the right ear and 1 o'clock in the left ear to 6 o'clock.

Exposure of the Stapes Footplate. There is a surprising variation in how much of the incudostapedial surgical anatomy is visible on exposing the middle ear. Sometimes barely the

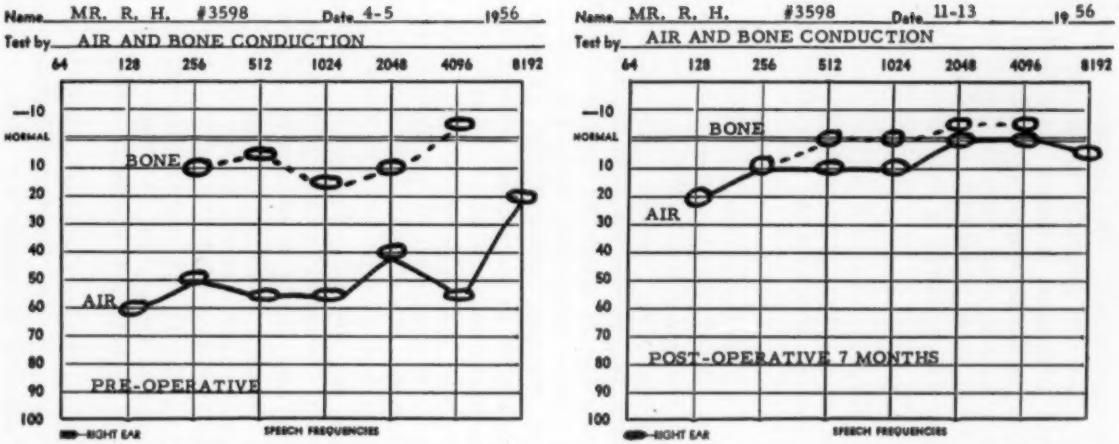


Fig. 8-B. Seven-month result showing that incudostapedial continuity has been maintained in incudostapedial joint disrupted during mobilization.

tip of the lenticular process of the incus is visible, and occasionally, most of the long process of the incus, the incudostapedial joint, the head, neck and part of both crura of the stapes, the stapedial tendon and some of the footplate can be seen simply by elevating the posterior half of the tympanic membrane. Since we have found that direct visualization of as much of the footplate as possible gives the best control of the mobilization procedure, we remove in most cases 2 or 3 mm. of posterior-superior bony rim (see Fig. 4) above the exit of the chorda tympani nerve to expose to view all of the stapedial tendon and its pyramidal eminence, the posterior crus, most of the footplate, and at times the anterior crus including the anterior margin of the footplate where it has been invaded by otosclerotic bone, as well as the long process and lenticular process of the incus, the capsule of the incudostapedial joint and head and neck of the stapes.. The bony canal of the facial nerve is very clearly seen just above the niche of the oval window, and in two of our cases there was no bony covering, the facial nerve being clothed only in the delicate mucoperiosteum of the middle ear. Depending upon its position, the chorda tympani nerve may be kept in its superior position, displaced downward, or if necessary, removed.

The operative work to this point has been done with the 2-X loupe magnification and the Lempert-Storz headlight.

Audiometry. Audiometric testing during the operation has been used by the authors from the beginning of our mobilization surgery as a helpful and valuable adjunct to digital and visual evaluation of the degree of mobility achieved. Since the objective of audiometric control is to demonstrate relative hearing gain obtained by adequate liberation of the ankylosed footplate the first hearing test is after the middle ear has been entered and the traumatized skin flap and drum membrane have been replaced. A sterilized sponge-rubber ring and cloth sleeve are used for the audiometer earpiece and cord (see Fig. 5) as devised by Rytzner[13] for his measurements of hearing changes during fenestration surgery. The four frequencies of 250, 500, 1000 and 2000 are tested.

Inspection of the Middle Ear. At this point in the surgery the Zeiss operating microscope protected by sterile drapes (see Fig. 6) is moved into position; the flap and drum membrane are re-elevated, and after careful positioning of the head and microscope for the optimum working angle, the incudostapedial joint, both crura, the footplate and the round

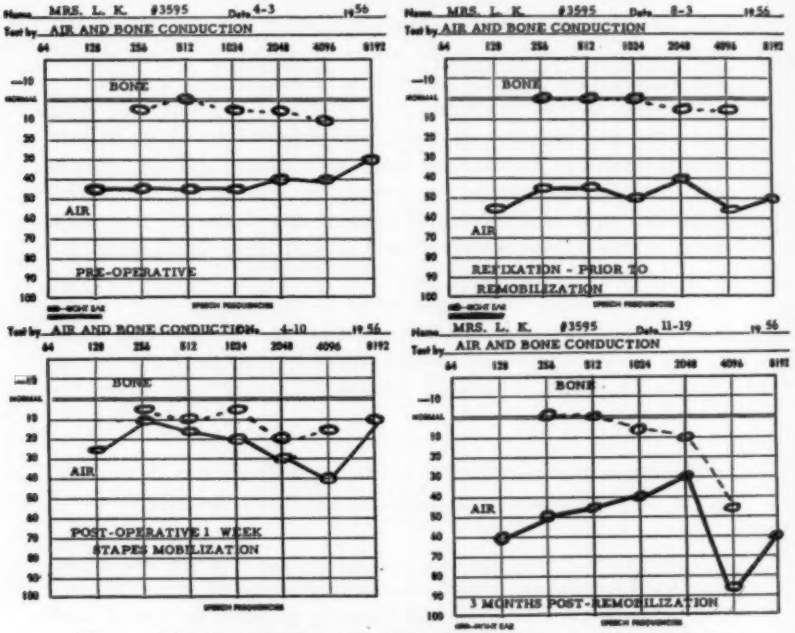
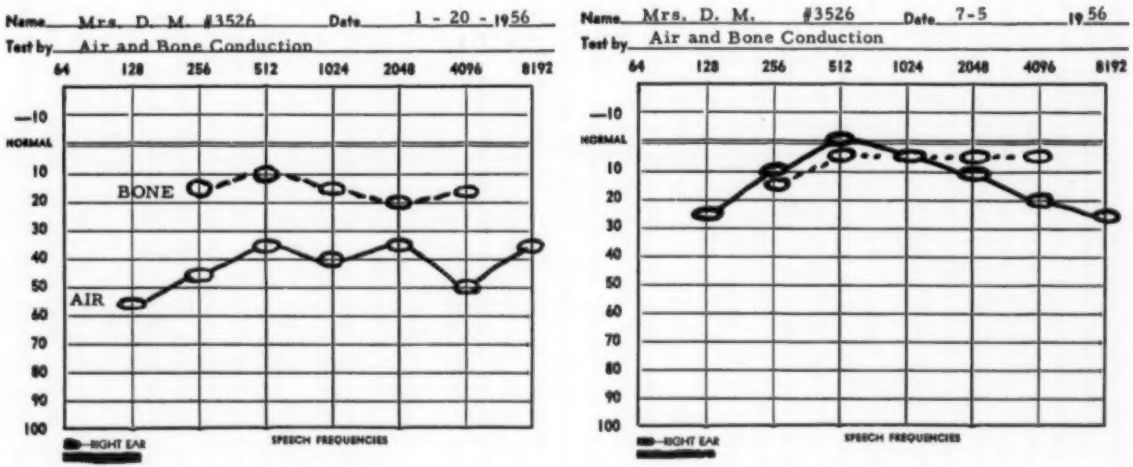


Fig. 9. Initial success following stapes mobilization in spite of fracture of both crura and dislocated incudostapedial joint. After refixation a remobilization gave an initial improvement up to 17 db. followed by rapid loss, with a lasting loss both in bone and air-conduction at 4096 cps.

window niche are minutely inspected under 16-X magnification. Very often delicate filmy adhesions in the niche of the oval window must be separated to gain full footplate visualization.

This preliminary inspection may reveal a footplate completely replaced by thick, chalky otosclerotic bone interlaced



Pre-operative. Post-operative 6 months.

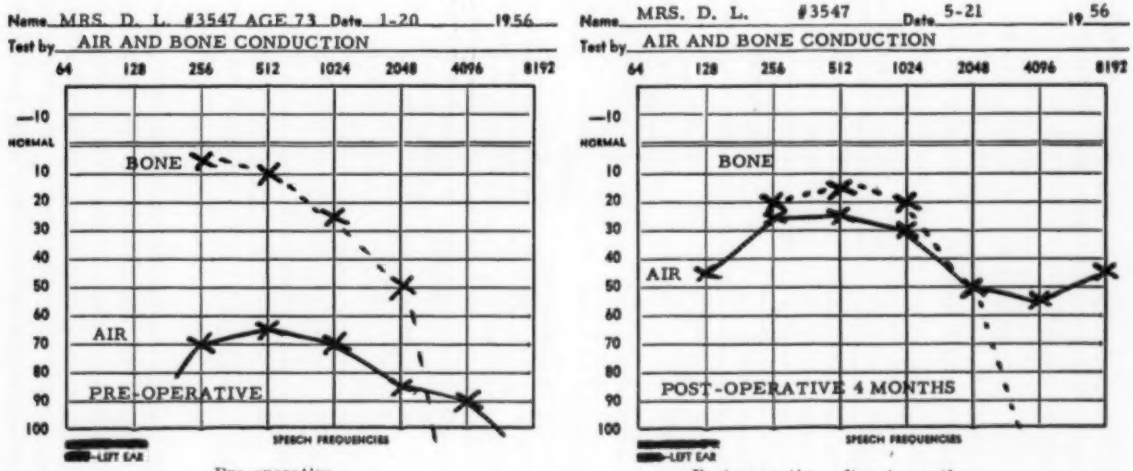
Fig. 10. Initially successful result of stapes mobilization. (Note reversal at Carhart notch).

with blood vessels of variable thickness, a situation which we have found is absolutely immune to successful mobilization. Should this patient be an "A" or "B" candidate for fenestration the operation is terminated by replacing the tympanic membrane and flap. In a case unsuitable for fenestration an attempt may be made to fracture the otosclerotic footplate with the House air-driven hammer,[14] but we are not yet convinced of the wisdom of this course.

As pointed out by Anson[15] the incus and malleus are relatively constant in form and structure, whereas the normal adult stapes shows extraordinary variability in the size, shape and strength of the crura and footplate. This extreme variability is borne out by preliminary inspection. When the crura are found to be unusually delicate, the anterior crus most often being so affected, the surgeon will be very cautious in his exploratory palpation and mobilization attempts. It is especially in these cases with delicate crura that the direction of force, introduced by one of us (E.L.D.) is more likely to succeed than previously described maneuvers. If such a stapes does not mobilize readily it may be advisable to loosen or free the margin of the footplate superiorly, anteriorly and inferiorly with the microscopic 10° hook (see Fig. 4), or with special tiny angulated chisels (see Fig. 7) prior to application of further pressure.

Following careful inspection, the incus, incudostapedial joint and stapes head are gently palpated with the microscopic 10° hook. In cases of early partial otosclerotic fixation slight pressure in an antero-posterior, vertical or oblique direction may evoke slight motility of the stapes, but as a rule the stapes is found to be rigid. Palpation also determines whether the incus and stapes head are solid or unusually soft, and whether the incudostapedial joint capsule is loose or tight. This helps to determine whether the mobilizing force will be applied through the incus or directly to the head of the stapes.

Direction and Application of Force in Mobilization. Just as the creation of the fenestra is the crucial stage in fenestration surgery, so the application of force for mobilization is the crucial step in the preliminary operation for stapes



Pre-operative.

Post-operative after 4 months.

Fig. 11. Successful mobilization with post-operative air within 10 decibels of pre-operative bone.

mobilization. It is this part of the procedure that is the most difficult to master, and, therefore, we shall describe it in considerable detail.

The stapedial crura come together at the neck in either a pointed or a rounded arch, and, as in any arch, the structure has its greatest strength against a force applied through the

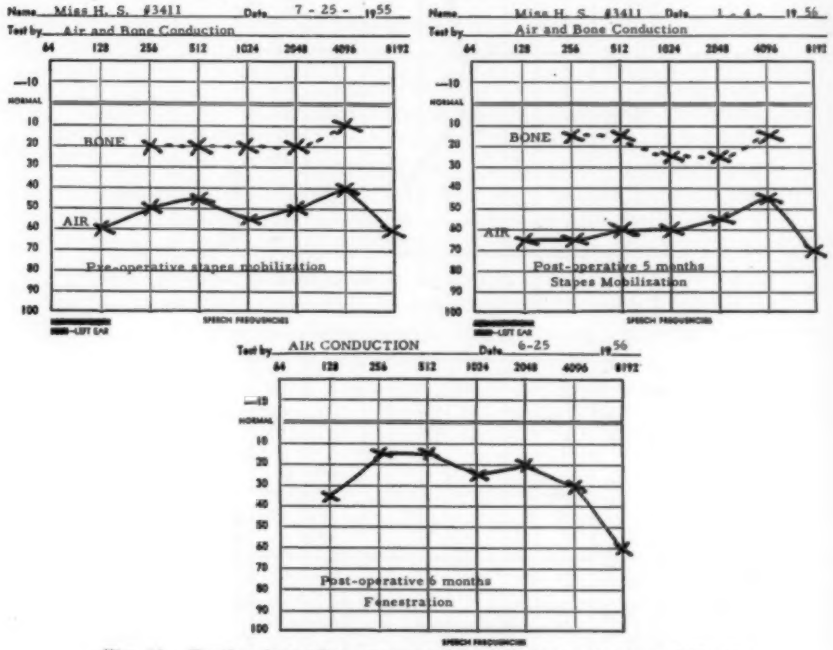


Fig. 12. Further loss after unsuccessful preliminary stapes mobilization, with good result from subsequent fenestration.

apex directly downward against the base, or in the case of the stapes, directly inward toward the footplate and vestibule. This mechanical and architectural principle was first clearly applied to mobilization by one of us (E.L.D.). Since adopting this as the *chief direction of force* to mobilize the footplate, our successful mobilizations with good audiometric

improvements on the operating table have increased. Cases which show beginning partial "greenstick" fracture of the crura during the application of antero-posterior, vertical or oblique force may still allow successful mobilization of the footplate in some cases, without completing the crural fracture, by direct inward pressure.

It is very important that the pressure be applied *intermittently* in a pulsating manner as advocated by Rosen. The force should be increased only very slowly and gradually. One of the hardest things to learn is that patient persistence in continuing an intermittent pulsating pressure of moderate force over a period of time will often succeed in freeing a rigid footplate when an impatient rapid increase in force will crush the crura beyond repair. Surgeons who garden as a hobby will find that exactly the same principle may be applied to pulling large weeds; an intermittent gentle tug applied chiefly in the axis of the stem directly outward, but alternating with pressures in other directions, slowly and cautiously increasing the force, will finally avulse the entire weed with its roots intact when a quick application of force will break it off. Rosen[16] makes a good suggestion; when the surgeon finds himself becoming tense he should leave the operating table and walk about to relax for a few moments before resuming his cautious and patient mobilization attempts.

Since the sharp pointed mobilizer tends to split or shear off part of the incus or stapes head, one of us (E.L.D.) devised a flattened end mobilizer with a roughened surface to prevent slipping (see Fig. 4). In some cases the sharp pointed (Shambaugh) mobilizer seems to work out, but in more cases the blunt (Derlacki) mobilizer seems most satisfactory for inward pressure.

If the incudostapedial joint is very loose, or if the lenticular process of the incus fractures, the inward force is applied directly to the head of the stapes after incising the joint capsule posteriorly with a tiny angled knife (see Fig. 7). Occasionally, the head and neck of the stapes fracture, but the footplate sometimes may still be mobilized by inward

force applied against one of the crura near the apex of the crural arch.

When inward pulsating pressure continued patiently and with restraint for some time fails to gain any mobility, or when the mobility gained is insufficient, the application of force inward may be alternated with antero-posterior, vertical, or oblique force exerted through the pointed mobilizer inserted into the incus or head of the stapes.

Continued intermittent pulsating pressure may result in a very slow gradually increasing mobility, or in a sudden "give" followed by free mobility of the footplate. In some cases the footplate as viewed through the operating microscope can then be seen to move well posteriorly while the anterior end is still attached as though by a hinge. Audiometric testing with the drum membrane and flap replaced may show only a slight hearing gain. By applying further intermittent pressure in an inward direction but chiefly toward the anterior crus, the anterior end of the footplate is finally liberated, or, in some cases, the ankylosed anterior crus fractures. The entire footplate may now be seen to move freely with the slightest pressure on the incus, and the hearing gain may reach 30, 40, or even 50 db. for individual frequencies in cases with a large preoperative air-bone gap.

With 16-X magnification not only the precise degree of footplate mobility, but also the exact pattern of any footplate fracture may be seen and recorded. Bleeding or perilymph escape from the footplate is noted, and the final position of the stapes relative to the oval window and incus is observed. This becomes particularly important when vigorous force application was necessary to mobilize a firmly ankylosed footplate, as a result of which the incudostapedial joint was disrupted, or the mobilizing force caused a slight displacement of the stapes into the labyrinth. In such cases various placements of the stapes may show a surprising variation in the hearing threshold (see Fig. 8), it being necessary to do repeated audiometric testing to select the most favorable position for the stapes in the oval window and against the incus.

In the event of crural fracture the surgeon may anticipate a failure, and often an appreciable further hearing loss resulting from disruption of a still-slightly-functioning ossicular chain; however, the crural fragments should be approximated, since very occasionally a footplate fracture occurs simultaneously with crural fracture and such a patient may still obtain a good result. Theoretically, it is doubtful that fractured crura will heal by bony union, since they consist of endochondral bone, and we have observed very definite fibrous union in several cases inspected six months later at the time of fenestration; however, we now have one case in which a remobilization attempt four months after the original stapes mobilization revealed a bony healing of an anterior crus fractured and dislocated at mid-level as well as capsular healing of a torn and dislocated incudostapedial joint (see Fig. 9).

When the surgeon has attained what he considers adequate mobility (and we strive for greater mobility now than in our earlier operations), all blood is aspirated from the middle ear, including the niches of the oval and round windows, the meatal flap, and tympanic membrane are replaced, and the hearing is tested. If there is a large hearing improvement of 30 or more db. for individual frequencies no further manipulation is indicated. If the hearing gain is less, the surgeon may wish to try for further increase in mobility of the stapes or repositioning of the mobilized stapes.

Packing and After-Care. The meatal flap is held snugly but not tightly by small cotton ball packs moistened in gantrisin otic solution placed against a strip of surgical rayon to facilitate their removal. Should there be a perforation of the drum membrane, a sterile cigaret paper disc should be placed over it as a splint, and allowed to separate by itself a few weeks later.

Postoperative care is minimal in comparison to that of the post-fenestration patient. Vertigo for a few hours postoperatively may follow mobilization with escape of perilymph, and may require Dramamine (R) or a similar drug. Unless

there has been bleeding into the labyrinth from fracture of the footplate, vertigo is rarely experienced after the day of operation. An antibiotic and sulfonamide in combination are given four times daily for five days as a prophylactic chemotherapy. Penicillin or other antibiotic is withheld unless there is evidence of postoperative infection.

The patient returns home the day following surgery and may then return to work. At one week the cotton ball packs are removed, using the same sterile dressing technique as in post-fenestration dressings.[9] The drum membrane and flap will vary in appearance at this time from near normal to a still bruised look with hemotympanum. At the second postoperative visit at one month the drum membrane and canal appear quite normal.

The hearing, frequently improved at one week, generally reaches its maximum at one month, but in some cases it continues to improve for several months, while in others it shows a decline. Refixation, with loss of the improvement, is most apt to occur during the first three months, but may occur later.

Of the 477 stapes mobilization operations performed by the authors, 440 have been tested one month to 24 months after surgery and are included in this summary of results.

We have attempted to apply to stapes mobilization the same sort of rigid criteria for success or failure that we have previously applied to fenestration results. There is, however, this difference: experience in fenestration surgery has shown that fenestras rarely close later than two years after operation, so that the two-year test after fenestration may be considered the "final" result of this procedure. On the contrary, there are as yet no reliable data regarding a time limit beyond which the stapes is unlikely to refix. There is a definite possibility that after several years most of the surgically mobilized stapes will refix as the otosclerotic focus enlarges or the fractured footplate heals. Thus, any favor-

able results achieved by this new procedure must be considered as preliminary.

In this statistical study we consider as surgical successes all cases that were at or above the 30 db. limit of practical hearing at the most recent test one month to 24 months after operation. Cases are also included as successes when the last test was within 5 db. of the prediction for fenestration (Shambaugh formula), or when it exceeded this prediction by 10 db. (within 5 db. of eradication of the preoperative air-bone gap).

Cases are considered failures that failed to reach the 30 db. level and were within 10 db. of the preoperative level (no change); that were more than 10 db. below the preoperative level (worse), and that gained more than 10 db. but failed to come within 5 db. of the predicted result of fenestration.

Cases were tabulated in four groups: the first 54 operations in which Rosen's technique was large employed; the next 26 cases where a sharp pointed mobilizer was used for applying force in various directions according to the method

TABLE I—TECHNIQUE 1.

Force toward Posterior Crus with Rosen Mobilizer—54 Cases—Most Recent Test 1-25 Months Post-operative.

	Classification No. of Cases	A 15	B 14	C 25	All 54
Surgical Success 24%	30 db. level*	5 (33%)	1	2	8 (15%)
	Not to 30 db.				
	±5 db. to Fen. Pred.	0	1	3	4 (7%)
	Exceed Fen. P. by 10 db.	0	0	1	1 (2%)
Surgical Failure 76%	Gain more than 10 db. but not to Fen. Prediction	0	1	0	1 (2%)
	No change ±10 db.	10	10	18	38 (70%)
	Worse by more than 10 db.	0	1	1	2 (4%)
*Exceeded Fenestration Prediction by 10 db.		0	0	2	2 (4%)

of Kos; a third group of 48 cases where the procedure was controlled by direct visualization of the footplate under 16-X magnification; and the most recent group of 312 cases where, under direct visualization of the footplate, the blunt ended (Derlacki) mobilizer and the sharp pointed mobilizer were used, applying force in various directions but especially directly inward (Tables I to V).

TABLE II—TECHNIQUE 2.

Force Variable Direction with Rosen and Kos Mobilizers—26 Cases Most Recent Test 1-14 Months Post-operative.

	Classification No. of Cases	A 8	B 8	C 10	All 26
Surgical Success 39%	30 db. level*	4(50%)	3	1	8(31%)
	Not to 30 db. ±5 db. to Fen. Pred.	0	0	2	2 (8%)
	Exceed Fen. P. by 10 db.	0	0	0	0
Surgical Failure 61%	Gain more than 10 db. but not to Fen. Pred.	1	1	2	4(15%)
	No change ±10 db.	3	4	5	12(46%)
	Worse by more than 10 db.	0	0	0	0
*Exceeded Fenestration Prediction by 10 db.		1	1	1	3

TABLE III—TECHNIQUE 3.

Footplate Visualization X-16, with Modified Kos Mobilizer—48 Cases Most Recent Test 1-13 Months Post-operative.

	Classification No. of Cases	A 14	B 11	C 23	All 48
	30 db. level*	8(57%)	4	5	17(35½%)
Surgical Success 54½%	Not to 30 db. ±5 db. to Fen. Pred.	0	3	4	7(15%)
	Exceed Fen. P. by 10	0	0	2	2 (4%)
Surgical Failure 45½%	Gain more than 10 db. but not to Fen. Pred.	1	0	1	2 (4%)
	No change ±10 db.	4	3	10	17(35½%)
	Worse by more than 10 db.	1	1	1	3 (6%)
*Exceeded Fenestration Prediction by 10 db.		2	3	5	10(21%)

TABLE IV—TECHNIQUE 4.

Footplate Visualization X-16, Chisel and Derlacki and Kos Mobilizers
312 Cases—Most Recent Test 1-9 Months Post-operative.

	Classification No. of Cases	A 153	B 93	C 66	All 312
Surgical Success 56%	30 db. level*	96 (63%)	35	7	138 (44%)
	Not to 30 db. ± 5 db. to Fen. Pred.	5	14	13	32 (10%)
	Exceed Fen. P. by 10 db.	0	0	7	7 (2%)
Surgical Failure 44%	Gain more than 10 db. but not to Fen. Pred.	6	10	2	18 (6%)
	No change ±10 db.	42	28	36	106 (34%)
	Worse by more than 10 db.	4	6	1	11 (4%)
*Exceeded Fenestration Prediction by 10 db.		24	16	7	47 (15%)

TABLE V—TECHNIQUE 5.

Most Recent Group—118 Cases—Test 1-4 Months.

	Classification No. of Cases	A 59	B 35	C 24	All 118
Surgical Success 64%	30 db. level*	40 (68%)	15	2	57 (48%)
	Not to 30 db. ±5 db. to Fen. Pred.	3	6	7	16 (14%)
	Exceed Fen. P. by 10 db.	0	0	2	2 (2%)
Surgical Failure 36%	Gain more than 10 db. but not to Fen. Pred.	2	6	1	9 (8%)
	No change ±10 db.	14	8	12	34 (28%)
	Worse by more than 10 db.	0	0	0	0
*Exceeded Fenestration Prediction by 10 db.		14	6	2	22 (18%)

COMMENT.

The percentage of preliminary improvements became greater when the Rosen technique was modified by application of force in various directions, and it became still greater when the procedure was controlled by visualization of the footplate under 16-X magnification. We believe that the improved results achieved by the three surgeons are more

than would be expected from increased experience with this new procedure.

When the cases are classified into "A," "B," and "C" suitability, according to the criteria used to judge suitability for fenestration, the percentage of successful results is considerably higher in "A" and "B" cases than in cases of "C" suitability. We believe that this is because the cochlear nerve deterioration that determines the "C" classification is more frequently associated with large otosclerotic foci that often invade the entire footplate, rendering it immune to mobilization.

Especially noteworthy is the considerable number of cases with further losses of hearing. In the entire series of 440 operations, 16, or 3.5 per cent, showed a further loss exceeding 10 db. for the speech frequencies at the most recent test. Some of these are accounted for by fracture of the crura, increasing the conductive loss; but others are the result of cochlear damage, as evidenced by a drop in bone conduction acuity, due probably to hemorrhage into the labyrinth, or to concussion from use of the pneumatic hammer.

We call attention to the cases whose hearing result exceeded the predicted result of a fenestration by 10 db. or more. In the entire series there were 62 cases that reached or exceeded the 30 db. limit of practical hearing and, at the same time, exceeded the predicted result of fenestration by 10 db. or more. There were an additional 10 cases that did not reach the 30 db. level but did exceed the predicted fenestration result by 10 db., a total of 72 cases, or 16 per cent of all mobilization operations that showed a hearing result superior to what a fenestration might be expected to yield.

CONCLUSIONS.

Three surgeons have evolved a technique for stapes mobilization that is yielding a 30 db. or better level of hearing in 63 per cent of cases of "A" suitability for fenestration. Roughly half of all mobilizations in all classes of cases fail to achieve a hearing improvement comparable to fenestration, while 3.5 per cent of all mobilizations end up with a further hearing loss of significant degree.

These results are preliminary, and their permanence is entirely a matter of conjecture.

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PROTRUSION OF THE EARS.*

JOHN B. ERICH, M.D.,

Rochester, Minn.

Despite the fact that new operative techniques are being developed year after year for the correction of protruding ears, repair of this defect is not in a final stage of perfection, and there is much opportunity for improvement. A review of the methods of treatment which have been described in the literature discloses that the basic surgical principles for rectifying a protruding ear were outlined more than 40 years ago, and these principles are still in effect today. Many modifications of these basic procedures have been described, of course, and in recent years, numerous refinements in technique have been added.

Prior to 1910, the majority of operations for protruding ears involved the excision of an elliptical piece of skin and sometimes cartilage along the postauricular sulcus; usually, this ellipse of tissue came partially from the auricula and partly from the mastoid process. Although suturing of the wound will correct the protrusion, such operations tend to eliminate a large part of the postauricular sulcus, and this result is most undesirable. This method of surgical treatment was recommended by many of the early plastic surgeons, such as Monks,[1] Joseph[2] and Morestin.[3] Although several surgeons are credited with developing this particular type of procedure, it often is referred to, rightly or wrongly, as the Morestin technique.

LITERATURE.

In 1910, Luckett[4] observed that operations designed to change temporarily the cephalo-auricular angle of protruding ears neither restored the normal anatomy of the ear nor gave lasting results. He brought out the fact that in this type of

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deformity the antihelix (or anthelix) is underdeveloped or nonexistent, and also that the concavity of the concha is continuous with the fossa triangularis, since both crura of the antihelix are essentially absent. He was the first to describe the technique of excising from the posterior surface of the ear a crescentic piece of cartilage along the proposed site for the antihelix; in addition, a similarly shaped segment of skin also was removed from the posterior aspect of the auricle. Catgut sutures were buried in the perichondrium to fold the ear back in proper position and create a new antihelix. Speaking of the operative site, Luckett cautioned against infections which could result in a deformed, thickened or corrugated ear. If the auricle contained thin flexible cartilages, he stated that the antihelix might be reconstructed without excision or incision of the cartilage; treatment then would consist merely in folding the cartilage at the desired site and in placing sutures for proper retention.

Selfridge,[5] in 1918, reported that he followed Morestin's technique for correcting protruding ears. He excised an ellipse of skin on the back of the ear and adjacent mastoid region to expose the perichondrium and periosteum. He then sutured the perichondrium and periosteum together with catgut to anchor the ear to the side of the head.

Lockwood,[6] in 1929, also following Morestin's principle, recommended excision of crescentic sections of skin and cartilage and stitching the cut edges together with so-called Lembert sutures. When these stitches were tied, the ear was held firmly against the side of the head.

Davis,[7] in 1929, gave an historical outline of the otoplasties, including operations for protrusion of the ears.

Writing about protruding ears, Goodyear,[8] in 1933, described an operative procedure which involved the excision of an elliptical piece of skin from the posterior auricular sulcus, as in the Morestin operation. In addition, he excised two other elliptical pieces of skin from the back of the ear. Suturing of the latter two wounds helped to correct the protrusion by establishing a more normally contoured antihelix. In his operation, no cartilage was incised or removed, and

it seems doubtful whether the configuration of the newly formed ear was maintained for any great length of time.

Wolfe,[9] in 1936, outlined a modification of the Luckett procedure in which cartilage along the antihelix was removed through an incision on the posterior surface of the ear. If the ear protruded most prominently at the upper pole, the so-called heart-shaped incision of Kolle was considered desirable. If the protrusion was centrally located, an ovoid incision was thought best. He stressed excision of cartilage without button-holing the anterior surface of the skin. No buried stitches were employed; instead, Wolfe simply sutured the skin so that the scar was located along the posterior auricular sulcus. The depressions of the ear were packed with cotton, and a snug pressure dressing was applied.

Davis and Kitlowski[10] thoroughly discussed the problem of protruding ears, in 1937. They gave a detailed account of the embryologic development of the external ear and stated that the auricle forms during the sixth week of fetal life from six tubercles on the mandibular and hyoid arches and also from an elevation of skin behind the tubercles on the hyoid arch. The ear was said to assume definite form during the first part of the third month, but the antihelix does not become definitely folded, and the crura do not appear until the sixth fetal month. When completely developed, the ear is said to rest at an angle of about 30 degrees with the head.

These authors brought out the fact that the principal cause of prominent or protruding ears is the incomplete development of the antihelix. Although such defects usually are congenital in origin, it was thought that they occasionally could be acquired by young children who constantly sleep with ears curled over or who frequently have their ears rolled forward under carelessly placed caps. Several operative techniques were described which varied from simple excision of skin to more complicated procedures involving the cartilaginous framework of the ear. Excision of skin alone was relegated to cases of slight deformity in young children. Davis and Kitlowski considered that if lasting results are to be obtained, the spring of the cartilage must be broken, and the antihelix reconstructed. They mentioned several procedures which can

be used to overcome resistance or spring of the cartilage. One may thin out the cartilage, one may incise the cartilage, or one may excise crescentic or elliptical pieces of cartilage along the line of the antihelix.

Davis and Kitlowski described their usual technique, which was derived from the Luckett and Morestin procedures. An elliptical segment of skin was excised from the ear and side of the head. Penetrating needles were used to mark out the proposed site for the antihelix on the posterior surface of the auricular cartilages. This line guided the surgeon in excising an elliptical segment of cartilage along the full length of the antihelix; care was taken to make sure that all of the cartilage was divided so that no tension or resistance existed when the cut margins were turned in to form the new antihelix. Buried Lempert catgut sutures through the perichondrium were used to bring out the fold of the antihelix. They stressed the importance of applying a dressing which would thoroughly immobilize and support the newly adjusted cartilages of the ear. They stated that it is not possible to give specific directions concerning the exact amounts of cartilage and skin to be removed as they vary greatly from case to case.

Brown,[11] in 1938, recommended the excision of an elliptical section of skin from the posterior surface of the auricle. He stated that the cartilage in the region of the antihelix should be shaved as thin as possible but should not be completely incised because of the likelihood of an irregular or unnatural appearance to the cut edge of the cartilage. Otherwise, his technique was similar to that described by Luckett[4] and Davis and Kitlowski.[10]

MacCollum,[12] in 1938, wrote an interesting article in which he stated that of 180,744 children admitted to the Children's Hospital in Boston between 1918 and 1938, only 46 were treated for malformation of the ear, and of this number only 21 had protruding ears. He stated that the cause of lop or protruding ears is a congenital overgrowth of the conchal cartilage and lack of formation of the antihelix. In his opinion a repair is not satisfactory unless the angle between the ear and mastoid region is reduced at least to 30 degrees,

unless the convolutions of cartilage are shaped to form an antihelix, and unless the incision in the skin is hidden behind the ear so that it is not noticeable. He mentioned three surgical procedures for correcting protruding ears. The first technique (a combination of the Morestin and Luckett operations) involved excision of skin and cartilage, interrupted catgut sutures to bring out the antihelix and, in addition, the suturing of the cartilages to the adjacent fascia of the scalp. He stated that the disadvantage of this technique was obliteration of the posterior auricular sulcus. The second technique did not involve removal of any skin but did involve excision of cartilage and closure with horizontal mattress sutures of fine silk in which the cut cartilages were sutured back to back. His third technique was a combination of the two previously mentioned surgical procedures.

Fomon,[13] in 1939, discussed the problem of outstanding or protruding ears, and stated that the only corrective measure of permanent value involved a reduction in the size of the encephalo-auricular angle by removal of a crescent-shaped section of postauricular skin and cartilage designed to break the spring of the remaining cartilage. He said that the amount of cartilage to be removed can be determined as follows: Seat the patient before a mirror and press the ears against the head. The line of contact between the ear and head can be outlined with methylene blue, and this reveals the area of skin and cartilage to be excised. He listed three types of operations for protruding ears: *1.* simple excision of postauricular skin; *2.* excision of skin and cartilage; and *3.* fixation of the auricle to the mastoid process.

Foucar,[14] in 1940, presented a rather detailed classification of congenital abnormalities of the ear. Twelve types of congenital defects were described, one of which was prominent or protruding ears. He gave a rather detailed account of the embryology of the auricle and described four operative procedures to correct protruding ears. The first involved a simple elliptical excision of postauricular skin. The second consisted of an elliptical excision of skin and cartilage. Foucar criticized this method because it does not reconstruct the antihelix satisfactorily. The third procedure required

an elliptical skin incision and a surgically prepared, V-shaped groove on the posterior surface of the cartilage at the site of the antihelix; the wound was closed by means of interrupted stitches in the perichondrium of the folded cartilage. The fourth procedure called for a postauricular incision through skin and cartilage along the antihelix, after which the cut cartilages were buckled back on each other and overcorrected by interrupted catgut sutures. Foucar thought that the fourth method was the technique of choice. He reported one case.

New and Erich,[15] in the same year, reported on an operation which in its details was somewhat analogous to those devised earlier, especially those of Davis and Kitlowski and MacCollum, but which differed from other operations mainly in the manner of inserting sutures which formed or molded the antihelix.

Cox,[16] in 1941, reviewed the historical background for total reconstruction of the ear, and congenital and acquired defects. He stated that for protruding ears the simplest and best operation consisted of excision of an elliptical piece of skin and cartilage from the posterior surface of the ear (Morestin's procedure).

Baxter,[17] in 1941, discussed the psychologic effects of protruding ears in children. He stated that there are two types of prominent ears: in one the antihelix is well-formed, but the size and convexity of the concha are increased considerably; in the other the conchal cavity is normal but the antihelix, especially the crura, is developed incompletely. He discussed numerous operations, and emphasized the fact that correction is most easily obtained in the young child whose cartilages are soft and pliable. Baxter used modifications of both the Morestin and Luckett operations, and stated that the procedure employed should vary with the type of deformity. He was of the opinion that, if the concha is abnormally large and concave with a relatively well-formed antihelix, the best corrective procedure involves excision of an ellipse of skin and cartilage, suturing the distal margin of the cartilage to the galea with interrupted fine wire sutures; however, if the antihelix is poorly developed, he recom-

mended excision of skin and cartilage along the antihelix and insertion of fine wire mattress sutures to hold the cartilage in the desired position. For adults he favored excision of two small wedged-shaped strips of cartilage along the antihelix; furthermore, he emphazised his belief that steel wire offers less reaction and better retention than other suture materials.

Coe,[18] in 1942, stated that protrusion of the ears is due to an incomplete or partial absence of the antihelix and its fossa. The surgical technique which he described for correction involved the formation of an adequate antihelix. He worked entirely from the posterior surface of the ear and did not penetrate the anterior surface with needles. In the region of the antihelix he thinned the cartilage down to the anterior perichondrium. In some cases he used several parallel incisions about 1 mm. apart, and sometimes removed two or three strips of cartilage between these numerous parallel incisions. Chromic catgut was used to suture the cartilage and form the antihelix. Excess skin was excised when necessary. Coe emphasized the importance of maintaining hemostasis and avoiding hematoma formation, which he said could organize and distort the auricle in a manner resembling a cauliflower ear.

Young,[19] in 1944, in discussing the problem of protruding ears, evaluated surgical methods of treatment and presented a modification of the Luckett technique. He recommended an incision on the posterior surface of the ear at the junction of the scapha and concha. This incision should extend through the entire length of the cartilage. He removed an ellipse of cartilage on the scaphal side superiorly and on the conchal side inferiorly. He then allowed the scapha of the ear to slide behind the conchal portion. The cartilage would remain in this position without retention sutures. He formed the triangular fossa by incising the cartilage along the inferior crus and the antihelix by the cut edge of the concha. According to Young, this operation will correct the abnormal protrusion and restore the normal configuration of the ear; furthermore, this technique does not produce the abnormal appearance which is given by an ear too close to the head.

Seeley,[20] in 1946, gave Young credit for the most logical

and practical method of correcting protruding ears. He described in detail a similar operation which not only permits formation of a well-developed triangular fossa but also allows for a reduction in size of abnormally large ears.

Weaver,[21] in 1947, discussed the whole problem of prominent or protruding ears, and gave special credit to New and Erich, Davis and Kitlowski, MacCollum and Luckett. The operation which he described is the same as presented by New and Erich in 1940.

Pierce, Klabunde and Bergeron,[22] in 1947, described a technique for the correction of protruding ears in which eight to ten parallel incisions were made almost through the cartilage in the region of the antihelix. Plain sutures of No. 4 catgut were used to hold the ear back and bring out the antihelix as desired. These authors stated that in their experience crosshatching or thinning the cartilage gave unsatisfactory results.

McEvitt,[23] in 1947, classified congenital deformities of the ear, and also thoroughly described the embryology of the auricle. He stated that attempts to suture the ear to the head on the theory that the whole structure is protruding will mutilate the concha and convert the postauricular sulcus into a triangular slit. Not only is the end-result abnormal in appearance but also often leads to irritation and infection. He divided protruding ears into three types: Type I consisted of ears of normal size with moderate protrusion caused by underdevelopment of the antihelix. Type II consisted of large ears with marked protrusion, underdevelopment of the antihelix and overprominence of the antitragus. Type III involved long narrow ears with little or no antihelix, the ear being curled forward so that it resembles a shell. McEvitt then proceeded to describe the various surgical techniques which have been devised for the correction of protruding ears and recommended the following treatment for the three types previously mentioned: For Type I, the skin should be excised with slight excision of cartilage along the antihelix and with no suturing of the cartilage. For Type II, considerable cartilage should be excised along the proposed antihelix with parallel incisions for weakening the

cartilage; the cartilage removed should extend well down into the antitragus, and the remaining antitragal cartilage should be thoroughly crosshatched. For Type III the cartilage should be weakened markedly and sometimes excised in all directions. To hold the cartilage in proper position, buried white silk sutures inserted through the perichondrium were recommended.

Franklyn,[24] in 1947, stated that the method of treatment most generally accepted for protruding ears is Joseph's technique in which an oval section of skin and cartilage is taken out from the posterior auricular sulcus. Franklyn described the objections to this procedure and recommended the removal of only a 2 mm. strip of cartilage. He stated that this plan had the advantage of simplifying the surgical technique in which only small amounts of skin and cartilage were excised. He claimed that the results of his technique were superior to older methods.

Seltzer,[25] in 1947, discussed the history of surgical procedures employed in the treatment of protruding ears and described a technique (modification of Morestin's technique) which he again discussed in another lengthy paper in 1954.[26]

Seltzer's method consisted of excision of an ellipse of skin from the posterior surface of the auricle and the mastoid process and also excision of an oval piece of cartilage; a comma-shaped portion of cartilage was left intact in the center for support of the conchal cavity. Subcutaneous catgut sutures were used for closure. Seltzer admitted that one objection to this technique was obliteration of the postauricular sulcus. He said, however, that this was objectionable only to barbers, not to the patient or the surgeon.

Rosedale,[27] in 1948, discussed congenital deformities of the external ear and described an operation for correction of protruding ears in which an elliptical piece of cartilage was removed for reconstruction of the antihelix (Luckett's technique).

Brown,[28] in the same year, also considered the problem of protruding ears and of changing the antihelix to more normal contour. His surgical technique was a modification of

Luckett's operation. After excision of an elliptical piece of cartilage along the proposed antihelix, Brown recommended sliding the cut edges of the cartilage over each other. He described in detail the dressing that he recommended be applied to the ears after the operation was completed.

Becker,[29] in 1949, went into detail on the embryology of the external ear and the etiologic factors of protrusion of the ear. In his opinion most of these cases are hereditary in nature, although in rare instances intrauterine injury may account for mild degrees of protrusion of the auricle, especially when the defect is unilateral. He discussed anatomic and pathologic features of this subject and gave a rather detailed history and résumé of the surgical techniques which have been designed to correct protruding ears. He rather favored Barsky's method, which produces a smooth and rounded superior crus, thus simulating a normal ear. In general, he followed the operations recommended by Luckett, Young, and Davis and Kitlowski.

Becker[30,31] wrote two more articles on protruding ears, one in 1950 and another in 1952. He compared the anatomy of the external ear of human beings with that of animals. He brought out the fact that removal of a strip of cartilage along the antihelix will leave an unnatural ridge along the surface of the auricle. In describing his operation, he emphasized the necessity of starting the excision of a diamond-shaped piece of cartilage near the antitragus and continuing upward to an apex opposite the sulcus antihelicis transversus. In addition to his operation for protruding ears, he considered protruding microtic lop ears. For this deformity, he recommended lengthening the helix by detaching the helix from the side of the head; subsequently, a flap of skin from the postauricular region would be rotated forward and inlaid between the side of the head and the cut edge of the helix. A free skin graft was applied to the donor site on the back of the ear.

Barsky,[32] in his book on plastic surgery published in 1950, described his operation for protruding ears which is based on the Luckett principles. His variation of the Luckett oper-

ation consisted of making two incisions along the course of the proposed antihelix. The intervening strip of cartilage was striated and not removed. He did, however, recommend excision of an elliptical piece of cartilage at the lower part of the conchal cavity and the trimming away of excess skin from the posterior surface of the ear.

Leonardo,[33] in 1950, presented his technique for the correction of protruding ears. His operation also is based largely on the Luckett procedure; however, Leonardo's technique stressed the importance of making the new antihelix extend into the superior crus rather than the inferior crus, as is the usual procedure. To him all internal sutures seemed unnecessary, and he merely closed the cutaneous wound.

Ragnell,[34] writing in 1952, stated that McEvitt's operation was satisfactory in the great majority of cases of protruding ears; however, in making the incision along the antihelix, Ragnell serrated the cartilage.

Vidaurre,[35] in 1952, stated that he had found McEvitt's and Luckett's procedures unsatisfactory. Instead, he recommended excision of an ellipse of skin and cartilage and use of catgut sutures to anchor the external cut edge of the cartilage occasionally behind the internal cut, but more often to the posterior surface of the conchal cartilage. He stated these sutures gave the necessary backward pull.

Gonzalez-Ulloa,[36] in 1952, reported that, in general, his operation for protruding ears resembled Luckett's procedure, but he made use of through-and-through filiform nonrusting wires to maintain the newly formed antihelix and superior crus.

Straith,[37] in 1953, wrote that in the reconstruction of the antihelix for protruding ears the crest of the newly formed antihelix should extend into the region of the posterior crus rather than into the region of the superior crus. He stated that in a normal ear there is a smooth curve along the crest of the antihelix into the inferior crus. In accordance with determinations described by Davis and Kitlowski, he corrected the height of the concha and antihelix. He emphasized

the importance of removing a strip of cartilage no more than 2 mm. wide.

OBJECTIVE OF THIS THESIS.

One phase of this presentation involves a study of the faults of an operation which another surgeon and I devised in 1940 for the correction of protruding ears. The main objective of this paper, however, concerns a description of

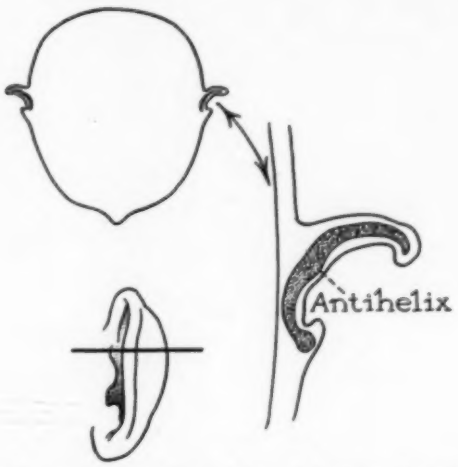


Fig. 1. Cross section of an outstanding ear illustrating the flattened antihelix and the resultant protrusion of the scaphoid and helical portions of the auricle.

certain definite improvements in the technique of this operation, improvements which the author has discovered through clinical study, surgical experimentation and review of the literature.

THE SURGICAL PROCEDURE.

The surgical procedure under consideration is primarily a modification of the Luckett operation. The technique is based on the assumption that an outstanding ear is the result of a congenital malformation of the antihelix (see Fig.

1). Normally, the contour of the antihelix is convex as viewed from its anterior surface (see Fig. 2), and it is this marked convexity of the cartilaginous antihelix that bends the scaphoid and helical portions of the ear inward toward the head, and prevents protrusion of the ear. It was the aim of the surgical technique being considered herein to restore the antihelix to its normal convexity (see Fig. 3). This, in turn, mechanically deflected the ear inward toward the head. Briefly, this was accomplished by incising the auricular

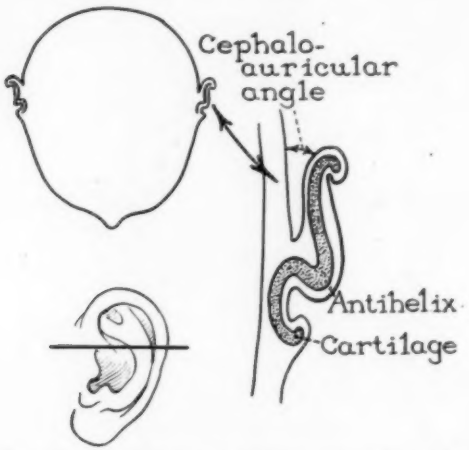


Fig. 2. Cross section of a normal ear illustrating the marked convexity of the antihelix.

cartilages in a vertical direction along the line of the antihelix, then by folding the cartilage and suturing the cut edges back to back, the protruding appearance of the ear was remedied.

A brief description of the operation under discussion, as it was originally designed, is as follows: Before the skin is incised, the proposed position for the antihelix is outlined with an indelible pencil or methylene blue on the anterior aspect of the ear. This line is carried into the inferior rather than the superior crus. Procaine or piperocaine hydro-

chloride (metycaine) containing epinephrine is injected subcutaneously both on the anterior and posterior auricular surfaces for hemostasis. An elliptical piece of skin which includes the sulcus formed by the junction of the auricle with the head is excised from the dorsal surface of the ear. At its greatest width this strip of skin measures about 1 cm. Adequate exposure of the dorsal surface of the auricular cartilages is obtained by undermining and reflecting the cutaneous margins of this wound. With the aid of a hypo-

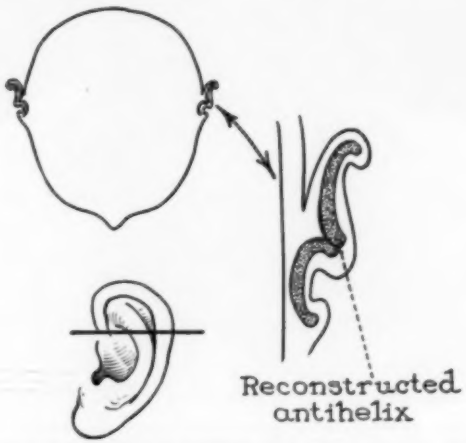


Fig. 3. Cross section of a reconstructed protruding ear. After excision an elliptical strip of cartilage, correctly inserted mattress sutures evert the cut edges of the cartilage to produce a convex antihelix, which, in turn, rectifies the abnormal state of protrusion.

dermic needle dipped in methylene blue, the position of the antihelix and inferior crus, as previously outlined in pencil on the ventral surface of the ear, can be transferred to the posterior surface of the cartilage (see Fig. 4). As a result of this process, a series of blue spots is produced on the exposed dorsal surface of the cartilage. This row of dots serves as a guide for the excision of an elliptical piece of cartilage which at its widest point does not exceed 5 mm. (see Figs. 4 and 5). A small scalpel and periosteal elevators are employed in removing the cartilage.

It is of extreme importance to make certain that the excised strip of cartilage extends the entire length of the ear and divides the auricular cartilages into two separate and distinct (mesial and lateral) parts. Even a small bridge of cartilage remaining between these two cartilaginous segments possesses an elastic force sufficient to restore the ear more or less completely to its original protruding position.

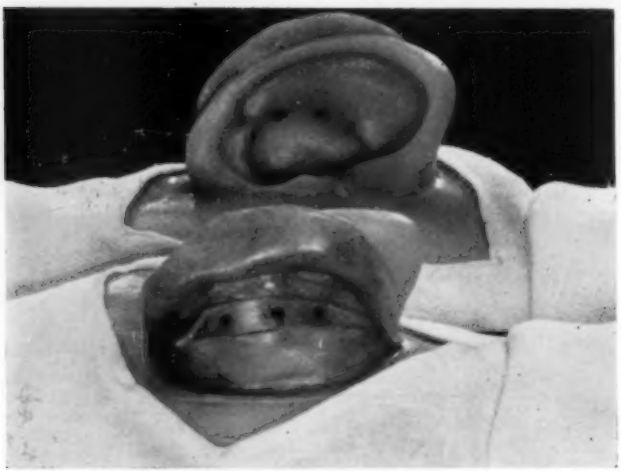


Fig. 4. Wax moulage of a protruding ear as seen from its posterior surface. The anterior auricular surface, which is visible, is a mirror image. In order to expose the cartilage, the skin is incised and reflected as illustrated. The series of dots marks the proposed position for the antihelix. One may note the outline of the elliptical strip of cartilage, which is being excised.

Mattress sutures are next inserted (see Fig. 5). Usually, four in each ear are required, and either white silk or cotton suture material is preferred because of its strength. Each mattress suture is introduced and also brought out through the anterior aspect of the ear, being inserted through skin and cartilage and across the cut edges of the cartilage posteriorly. The course of these sutures through the ear can be explained most easily by means of a diagram (see Fig. 5). Although inserted at this stage of the operation, these sutures are left untied until the cutaneous edges of the wound on

the posterior surface of the ear are carefully approximated with fine silk sutures. After closure of the posterior wound, the mattress sutures are tied firmly over small rolls of cotton (see Fig. 6). The tying of these sutures effects the following results: The cut edges of the cartilage are everted, and the cartilages near the cut edges brought back to back; this produces a convex antihelix, which, in turn, rotates the scaphoid and helical portions of the ear inward to correct the

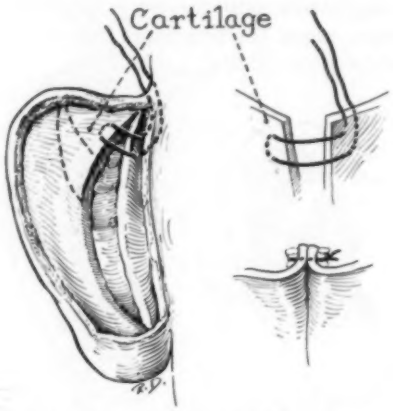


Fig. 5. The posterior surface of a protruding ear. The skin has been almost entirely removed to give a better view of the auricular cartilages. This drawing illustrates the elliptical strip of cartilage which has been removed, and the method of inserting mattress sutures from the anterior surface of the ear through skin and cartilage. When these sutures are tied over small rolls of cotton, the cut edges of the cartilage are everted to produce a convex antihelix. The dotted margins of the triangular area on the upper portion of the auricular cartilage indicate the site at which a secondary wedge of cartilage may be removed for the construction of a convex superior crus.

deformity. The amount of inward deflection of the ear is governed by the degree of tension placed on the mattress sutures when tied. Often it is desirable to expose the face and tie a mattress suture in each ear in rapid succession; this insures a symmetrical alignment of the two ears.

I believe that mattress sutures offer a more effective means of forming the antihelix than do subcutaneous catgut sutures. If the latter are employed, after the cutaneous

wounds have been sutured, there is no possible way of adjusting the position of the ear without opening the wound; furthermore, such catgut sutures occasionally are absorbed before the healing process is complete, and if an infection should occur the catgut will disintegrate when perhaps, its tension is most desired. The use of external mattress sutures obviates such complications.

ANALYSIS OF DEFECTS OF SURGICAL PROCEDURE.

While this operation will correct a protruding ear satisfactorily in many cases, the technique has, as stated previously, several conspicuous faults:

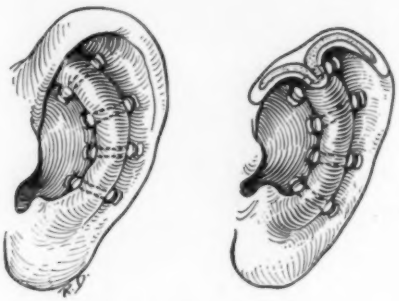


Fig. 6. The reconstructed ear with mattress sutures in position and tied over small rolls of cotton. A marked degree of convexity of the antihelix has been attained by this procedure.

1. The incision in the postauricular sulcus is often disagreeable for the patient.

2. Excision of an elliptical piece of cartilage is objectionable, because when the cut edges are everted and tied with mattress sutures, a sharp angulated antihelix is often produced which has an unnatural appearance.

3. When the reconstructed antihelix is extended into the inferior crus, as was described, it may not fully or permanently correct the deformity.

4. Mattress sutures when tied and inserted, as described,

often cause ulceration of the underlying skin which is most undesirable.

ALTERATIONS IN TECHNIQUE RESULTING FROM CLINICAL AND SURGICAL STUDY.

Through a clinical and surgical study, the author has found that the following alterations in technique will eliminate the faults just enumerated.

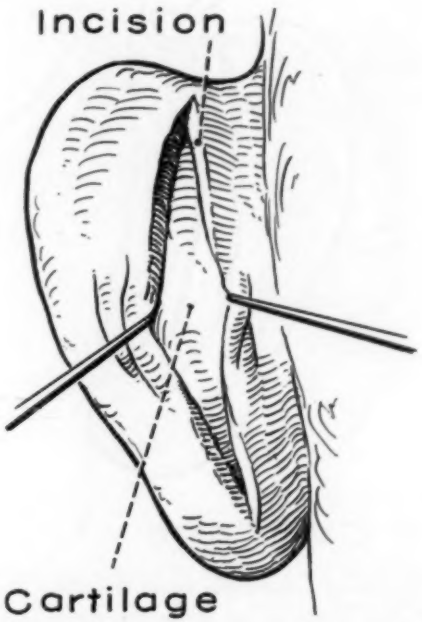


Fig. 7. Vertical incision which the author uses for protruding ears today. Incision is located over the region of the antihelix. If this incision is neatly sutured with fine silk, it is unnoticeable later on.

The Incision—Excision of an elliptical piece of skin from the posterior auricular surface of the ear is not necessary, as excision of such a segment of skin has no permanent value in holding the ear closer to the head; moreover, the post-auricular skin has enough elasticity to contract snugly over

the surgically repositioned cartilage, and the excision of a piece of skin only adds to the ultimate amount of scarring. The author has found that an incision in the skin in the post-auricular sulcus often will produce a scar which is tender and disagreeable to the patient who wears glasses with bows. To

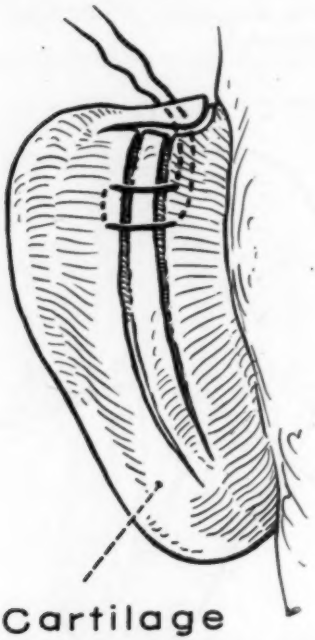


Fig. 8. Author's most recent method of incising the cartilage for protruding ears. The diagram shows the posterior surface of a protruding ear. The skin has been entirely removed. Two parallel incisions are made almost the full length of the ear in the region of the antihelix. The upper part of the helix is detached from the rest of the cartilages of the ear by a horizontal incision as is shown; this incision is carried upward to sever the continuity of the helix at the point where the helix joins the side of the head. See text regarding the advantages of these incisions in the cartilage over the older method shown in Fig. 5.

obviate this difficulty, the author has discovered that it is much more advantageous to make the skin incision on the posterior surface of the ear directly opposite the proposed position for the antihelix (see Fig. 7). Placing the incision

in this location not only eliminates the scarring in the postauricular sulcus but also allows a more direct surgical approach to the cartilages of the ear in the region of the antihelix. If the skin incision on the back of the ear is neatly sutured with fine silk stitches the resultant fine linear scar will not be noticeable.

Excision of Cartilage—Excision of an elliptical piece of cartilage to form the antihelix is definitely undesirable. When

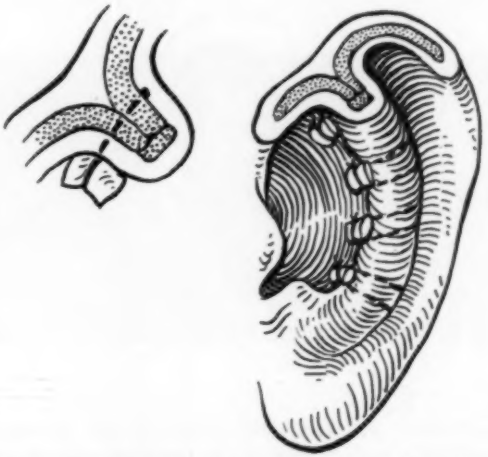


Fig. 9. The author's most recent method of inserting mattress sutures to bring out the antihelix and correct the protrusion of the ear. These sutures go through skin and cartilage mesial to the antihelix but only through the cartilage lateral to the antihelix; in other words, these mattress sutures are not visible lateral to the antihelix. They are tied over rolls of cotton on the mesial side of the antihelix and these rolls of cotton are all located within the concha of the ear. See text for advantages of this method of suturing the antihelix. The insert shows how the cut cartilages are brought back to back between mattress sutures to form the antihelix and correct the protrusion. Notice that the small strip of cartilage intervening between the two parallel incisions shown in Fig. 8 is pushed forward under the skin of the antihelix; this makes a much more rounded contour for the antihelix than is obtained through excising an ellipse of cartilage as is shown in Fig. 5.

the cut edges are everted and brought together by mattress sutures, a sharply angulated antihelix often is produced, which has an unnatural appearance. Many techniques have been considered and tried in an effort to prevent this sharply angular appearance. The method which I have found

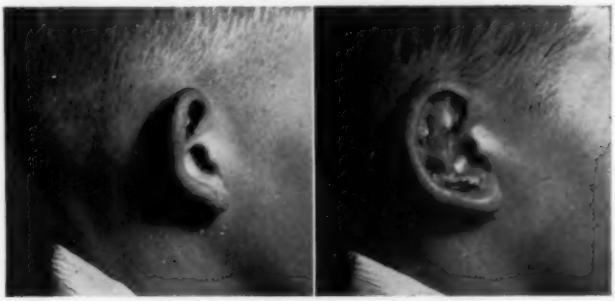
most effective and simplest involves the preparation of two parallel incision 2 or 3 mm. apart; these incisions are carried through the full thickness of the cartilage along the proposed site for the antihelix (see Fig. 8). The small strip of cartilage intervening between these two parallel incisions is left intact. When the mattress sutures are inserted and tied down to bring out the antihelix, this strip of cartilage is pushed forward to produce a well-rounded rather than an angulated antihelix (see Fig. 9).

Fig. 10. Older method of attempting to create a superior crus for the antihelix in an effort to prevent the scaphoid portion of the ear from lopping over after the plastic operation has been completed. See text.

Reconstruction of Antihelix—One of the greatest faults of the operation herein described involves reconstruction of the antihelix so that it runs into the site normally occupied by the inferior crus. Under this circumstance, the cartilages forming the scaphoid portion of the ear lack support and may lop over after the surgical procedure has been completed. Thus an undesirable secondary defect is created. Occasionally, this complication occurs at the time of operation, and no amount of tension on the mattress sutures will overcome the tendency of this part of the ear to hang limp and impair the final result. Years ago an attempt was made to correct this difficulty by accentuating the convexity of the superior crus. This was accomplished by removing a secondary small strip of cartilage in the region of the superior crus at the same time that the elliptical piece of cartilage

was excised along the line of the antihelix and inferior crus (see Fig. 10). While this technique is of some help in preventing the upper pole of the ear from lopping over, it does not always eliminate this complication.

I have found by trial and error that it is preferable to reconstruct the antihelix so that it extends upward into the superior crus rather than into the inferior crus (see Fig. 9). By so doing, the upper portion of the ear never lops over on completion of the operation; however, if the incisions in cartilage are carried into the superior crus, another compli-



a. b.

Fig. 11. a. Patient with protruding ears. b. Postoperative result 2 weeks later. Notice that small ulcers have developed in the skin lateral to the antihelix at the site of the mattress sutures. Such ulcerations heal without difficulty but sometimes produce scarring. The antihelix has been made to join the superior crus rather than the inferior crus.

cation occurs. As previously mentioned, the incision in the region of the antihelix must completely divide the auricular cartilage into two separate and distinct mesial and lateral parts. If the incision of cartilage is carried along the superior crus and across the helix, a bad defect in the form of angular buckling of the upper edge of the helix is produced when the mattress sutures subsequently are tied. Although the helix must be completely cut across in order to correct protrusion of the ear, I have found that the helix need not be divided immediately above the superior crus. Instead, the upper portion of the helix can be separated from the rest of the auricular cartilages by a horizontal incision that ex-

a. b. c.

Fig. 12. a. Patient with protruding ears. b. Mattress sutures have been inserted in accordance with the older method, and tied over rolls of cotton on each side of the reconstructed antihelix. c. Two weeks later. Mattress sutures removed. Notice the ulceration in the skin lateral to the antihelix. This ulceration can be avoided by the newer method of inserting mattress sutures.

tends mesially to the region where the helix joins the side of the head; at this particular site the helix can be cut across (see Fig. 8). This cut, in effect, interrupts the continuity of the helix about a centimeter mesial to the superior crus of the antihelix. This method of dividing the helix entirely eliminates the spring in this structure; moreover, when the mattress sutures are tied securely to evert the cut edges of the antihelix and superior crus, there is no visible buckling of the helix itself. As a matter of fact, the helix maintains a smooth normal contour but has no elasticity and no tendency to cause the ear to lop over or protrude.

Placement of Mattress Sutures—The final fault of the operation which I have described concerns the mattress sutures. Mattress sutures, however, when left in place for two weeks, may produce some ulceration of the underlying skin. These ulcerations heal without any difficulty but may leave small scars, some of which are conspicuous (see Figs. 11 and 12).

This undesirable complication can be avoided by inserting the mattress sutures in such a way that they pass through only cartilage on the lateral side of the parallel antihelical incisions, but through both cartilage and skin on the mesial side (see Fig. 9). In other words, these sutures in the lateral piece of cartilage are located below the skin over the ventral surface of the ear. Consequently, when tied, the mattress sutures can leave no scarring lateral to the newly formed antihelix. These sutures may produce some scarring on the mesial side, but since the scars are located in the concha, they are invisible except on close inspection of the ears (see Fig. 13). Insertion of mattress sutures in this manner still allows one to fold the auricle backward to any desired degree as a final step in the operation, but eliminates all scarring lateral to the antihelix.

SUMMARY.

The surgical correction of protruding ears is still not in a stage of complete perfection; however, in recent years, many surgeons have made definite contributions which tend to eliminate the faults of earlier methods. In this paper I have

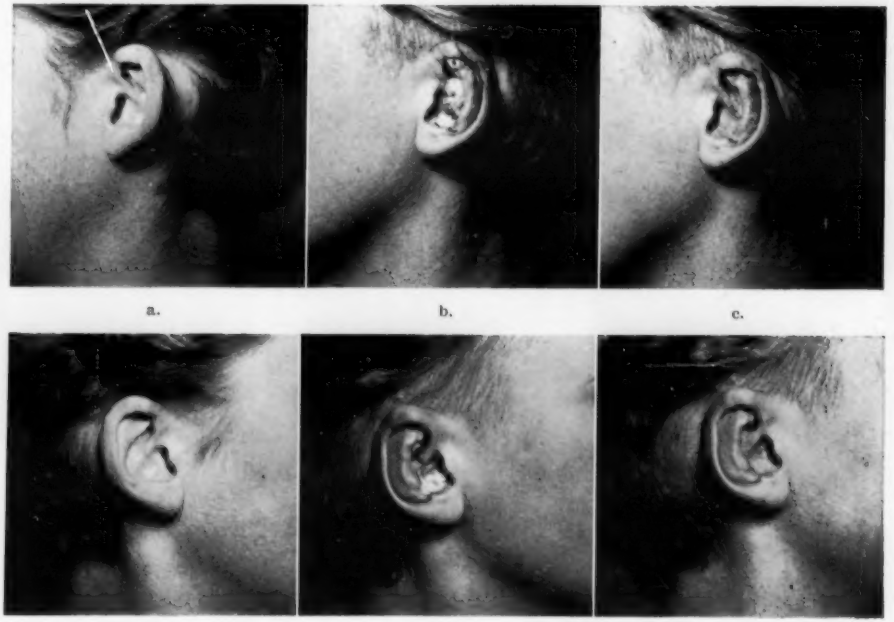
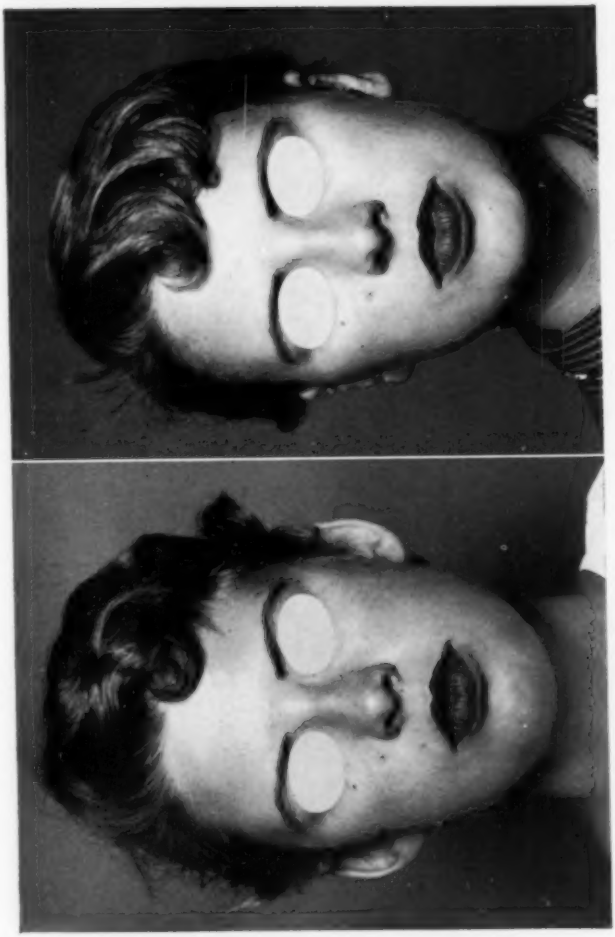
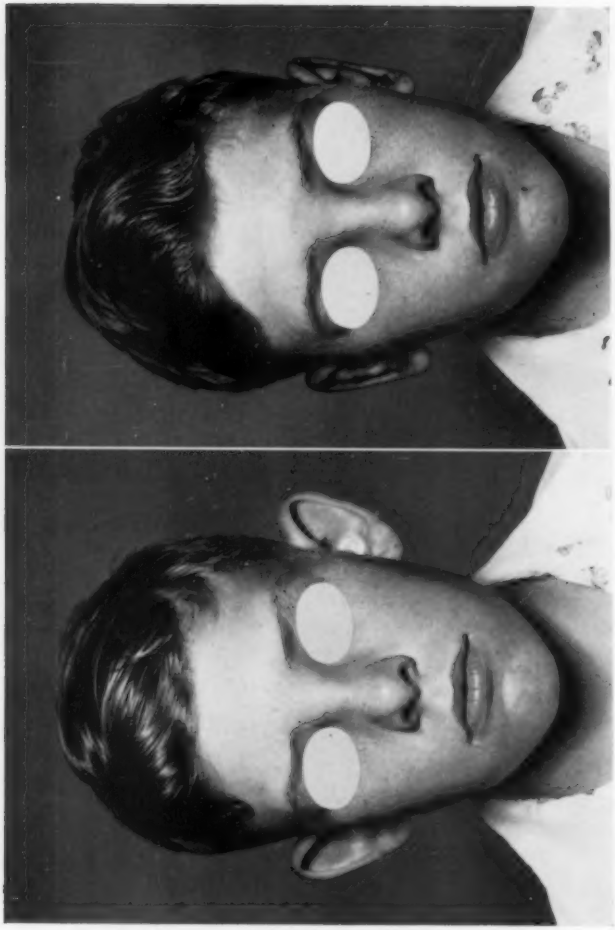


Fig. 13. a. Patient with protruding ear of left side. b. New antihelix reconstructed on left side; protrusion corrected. Notice that there are no rolls of cotton and that the mattress sutures are not visible lateral to the antihelix. c. Final result. Notice the contour of the antihelix. d, e and f. Same patient, right side. e. Antihelix reconstructed; protrusion corrected; mattress sutures in place. f. Final result.



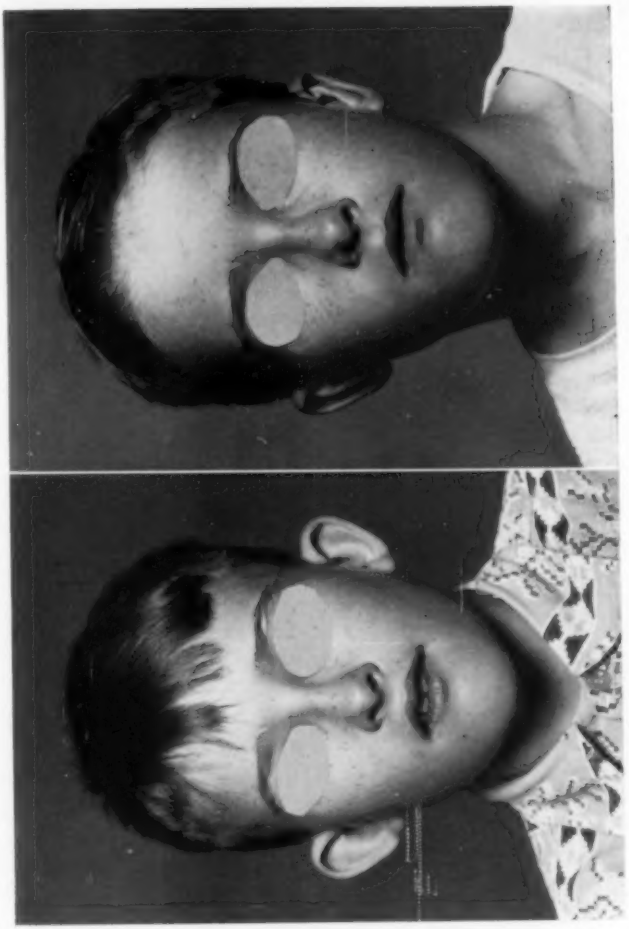
a. b.

Fig. 14. a. Patient with protruding ears. b. Deformity corrected by method described in the text.



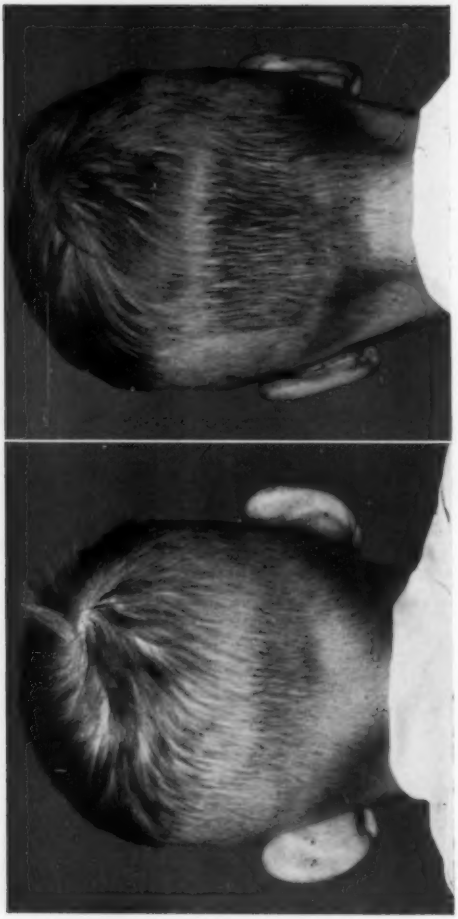
a. b.

Fig. 15. a. Patient with protruding ears. b. Deformity corrected by method described in the text.



a. b.

Fig. 16. a. Patient with protruding ears. b. Deformity corrected by method described in the text.



a. b.

Fig. 17. a. Patient with protruding ears. b. Deformity corrected by method described in the text.

attempted to review the imperfections of an operation for protruding ears which I described and used several years ago, and have presented the results of a clinical study designed to improve the end-results of this operative procedure by making certain changes in surgical technique (see Figs. 14 to 17).

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ALLERGIC VASOMOTOR RHINITIS.*

JACK V. TREYNOR, M.D.,

Council Bluffs, Iowa.

By personal experience as doctor and patient, the author has had intimate contact with the disease under discussion. This thesis may subconsciously represent an account of his conversion from extreme skepticism to sustained optimism.

It will be his purpose to show that vigorous attack on the disease, using all available methods in their proper places, can and does result in a high degree of patient satisfaction and comfort.

HISTORY.

When in 1565 Leonhardus Botallus[1] described a nasal reaction to the odor of roses, he undoubtedly was responsible for the term "rose fever," which has persisted until our own day. It would seem unfair, however, to blame him alone for all the ensuing confusion of terms and treatment that have plagued us since. Just as Botallus[1] seems to have been the first to have been keenly aware of a logical basis for nasal allergy, Van Helmont,[2] in about 1607, gave us what was probably the first description of pollen-caused asthma and, in 1698, Sir John Floyer[3] described asthma occurring only in Summer. An awareness of hypersensitiveness in asthma was shown by Bree[4] in his description of nasal and bronchial reaction to hair powder.

The outstanding landmark in nasal allergy is the work of John Bostock.[5,6] In 1819, he described, in minute and accurate detail, his own nasal condition and used the term "Hay Fever" in his discussions. Bostock's work was resumed by Blackley[7] in 1856, and by investigations which would be considered admirable by present standards, was able to prove the

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implication of grass pollens in hay fever. He could produce typical nasal symptoms consistently and at will by introducing identified pollen into the nose. More important, by rubbing pollen into the skin, he could produce reactions which must have been the first formally conducted skin test for pollen sensitivity. Further, Blackley was aware of mold spores and their relation to allergy, though the term was unknown to him.

Almost half a century elapsed before investigations again were focussed on pollens and molds as causes of respiratory allergy. In the interval, new theories arose: Von Helmholtz and Binz,[8] having found vibrios in nasal secretions of hay fever sufferers, were convinced that they had identified the cause of hay fever. Cessation of symptoms, seemingly due to the local use of quinine, and the subsequent disappearance of vibrios, further convinced these authors of the correctness of their theory.

A little later, in 1881, Daly[9] deduced that hay fever was due to pathologically sensitive areas within the nose. That his theory was acceptable to the practitioner of that day is attested by the volume of needless surgery performed on the nose thereafter.

Dunbar's work,[10] early in the present Century, should probably be credited with stemming the tide of empirical treatment of the allergic nose. By repeating and confirming Blackley's experiments, he placed the etiology of nasal allergy on a firm basis. Unfortunately, his efforts to treat hay fever with his "Pollantin"—an antitoxin derived from the blood of horses injected with pollens—almost nullified the gains from his researches. Almost simultaneously Weichardt developed "Graminol," which was a serum obtained from the blood of animals feeding on grasses. Acceptance and use of these agents by the profession undoubtedly delayed further a more logical attack on nasal allergy. Whereas Dunbar's work undoubtedly redirected investigation into channels which we now recognize as proper, it also delayed for some time a profitable attack on treatment.

Another half century elapsed between the two first recog-

nized works on experimental anaphylaxis. Magendie,[11] in 1839 injected dogs repeatedly with egg albumin. After a varying number of injections, death would be precipitated. Flexner[12] in 1894 was able to sensitize dogs to serum by a single dose and to cause death by administration of a second.

On the basis of the experiments of Magendie,[11], Flexner,[12] Hericourt and Richet,[13] the latter coined the term "anaphylaxis". About the same time (1903) Arthus[14] described the phenomenon which since has carried his name: severe, sterile local reaction at the site of injection of material to which an animal previously has been sensitized.

Two years later (1905) Von Pirquet and Schick[15] named and described "serum sickness". They showed that the chances of its occurrence were much greater in children previously inoculated with the same serum.

There must have been a surge of interest in the allergy question during the first decade of this century. In 1907, Otto[16] was able to demonstrate the role of antibody in anaphylaxis. By injection of serum from a sensitized guinea pig to a nonsensitive pig ("passive transfer") he was able to sensitize the latter. As a complement to the work of Otto, Besredka and Steinhart,[17] among many others, showed that an animal could be desensitized by serial injections of the antigen.

In 1906 the term "allergy" first appeared in Von Pirquet's[18] report of his work on tuberculin sensitivity. In 1906, Wolff-Eisner[19] identified hay fever as related to anaphylaxis, and in 1910 Melzer[20] similarly classified asthma. As a climax to the brilliant work of the previous decade, Noon and Freeman,[21] in 1911, brought out their classic report on pollen desensitization.

From this volume of work it was inevitable that curiosity as to the basic mechanisms of allergy must arise. Acceptance of the antigen-antibody reaction became fairly universal, but more specific bio-physical and chemical explanations were urgently sought.

The first acceptable study in this direction was by Dale

and Laidlaw[22] in 1911. They showed that histamine, which is derived from universally present histidine, when injected, caused anaphylaxis-like reactions. Wells[23] attempted to desensitize animals to histamine. Because of its failure to desensitize and to produce other changes typical of anaphylaxis in desensitized tissue, Wells discounted the relation of histamine to allergy; however, histamine was found by Lewis and Grant[24] in urticarial wheals due to stroking. Lewis deduced that in anaphylaxis there was an antigen-antibody reaction resulting in elaboration of an "H substance" which resembled histamine or was identical with it and caused dilatation and increased permeability of capillaries.

Best[25] devised methods for recovery of histamine from tissue and measured the amounts recoverable from various tissues. It is interesting that lung and epidermis contained far more recoverable histamine than did tissues not known to take part in allergic reactions.

Dragstedt and Mead[26] and, later, Code[27] showed marked increase in blood histamine after anaphylactic shock. On the basis of this work it was postulated by Dragstedt that allergy could be controlled by:

1. Reduction of histamines available for liberation.

2. Prevention of liberation of histamine.

3. Inactivation of liberated histamine.

4. Decrease of the effects of histamine.

This might well serve as an outline of our present concept of treatment.

Hyposensitization.

The next great step in control of allergy was in accord with the second of these postulates, when hyposensitization by injection of offending allergens began to take its rightful place as one of the basically sound approaches to allergic treatment. Less well known was the attempt at immunization against histamine proper by injection (into laboratory animals) of histamine azo-protein by Fell[28] and others. Although

some degree of protection was developed, it was so slight as to be impracticable, and the work was abandoned. Later, Horton's work[29] on histamine induced vasomotor disturbances indicated that some degree of lessened sensitivity to the substance may be obtained.

It is doubtful that complete immunity against histamine or against allergens implicated in vasomotor rhinitis has ever been attained; however, evidence has been accumulating over the past several decades that hyposensitization, or relative immunity, can be maintained by systematic and persistent injection of allergens. Two major methods have developed, each of which has its vigorous advocates. Rinkle[30] recommends *subcutaneous* injection of an amount of allergen extract which will produce a local reaction "not over 1½ inches in diameter and which is gone in eight to 18 hours," "this reaction varying with the adiposity at the site of injection." He also says that "wheal size is not as accurate as titration" in gauging dosage.

Opposed to this method is that of "optimum dosage" as practiced by Hansel,[31] Prewitt[32] and others. Here the injection is *intradermal* and the amount used minute, almost infinitesimal, and once the most effective dosage has been determined by clinical trial, that dosage is used without the progressive increase which other methods require.

The interval between injections has varied between wide limits in both methods, and both have incorporated the simultaneous use of histamine injections and nonspecific bacterial preparations on the theory that, to the nonspecific injection (and perhaps to the specific) there is an autonomic response comparable to that induced by steroids (Prewitt[32]).

Both methods must have real value. Perhaps their effects on the recipient are basically different, the high dose method producing true refractoriness to antigen and the low dose method an improvement in general autonomic stability. Both have contributed enormously to the treatment of allergic vasomotor rhinitis by opening a broad avenue of physiological approach.

Nasal Vasoconstrictors.

Around the turn of the Century the advent of cocaine opened a new chapter in the treatment of allergic rhinitis. Because it combined mucosal shrinkage with a high degree of local anesthesia, the drug met with enthusiastic response; so much so that cocaine addiction became widespread. Because of this factor and because of its very real danger in patients sensitized to the drug, its general use in treatment has been abandoned.

Epinephrine, long recognized as the most potent of the nasal vasoconstrictors, has commendably been withheld from general use by patients, especially since the appearance of ephedrine in the early Twenties. Since its advent, the latter has been available as an alkaloid and sulphate for use topically and enterally. Were it not for the side effects of cerebral stimulation and excessive pressor reaction it would still represent the ideal vasoconstrictor.

Many attempts have been made to approximate the sympathomimetic effect of ephedrine while reducing or eliminating its undesirable side effects. A host of clinically related substances have been synthesized, most of them efficient, all producing rebound in some degree except perhaps Tyzine (Tetrahydrozoline) which certainly has about as wide a vasoconstrictor-rebound latitude as any. The nasal mucosa seems to develop a progressive tolerance or refractoriness toward all of these drugs and increasing amounts seem necessary to satisfy the patient's demand for an open airway. With all possible good intent, the drug houses have met this addiction by marketing their "nose drops" as "nose washes" in plastic dispensers, and offer products which may be vehicles for sulfas, antibiotics, antihistamines, steroids and detergents singly and in combination. If these added agents caused no other ill effects, they might result in sensitization. Within the last few years the use of these drugs in inhalers has become popularized. Whereas, the overuse of "nose drops" was becoming a serious problem, the habitual, almost reflex, use of "sniffers" has become a social vice.

Antihistamines.

Dragsted's third postulate: "Inactivation of liberated histamine", has led to a search for substances which might do just that. According to Waters[33] as soon as there is an increase in concentration of free histamin in the extracellular fluids, this excess is attached to the protein molecules of the cells and then rendered inactive. At least part of the inactivating process is probably accomplished by the histaminase content of certain cells.

In search for this natural inactivator, Best and McHenry[34] isolated histaminase, which was later placed on the market as "Torantil" (not by the above investigators and not sponsored or endorsed by either). After an enthusiastic reception and a brief trial, no more was heard of the preparation.

Beginning in 1937, and continuing until the present, there has been a very active investigation of various drugs which have antihistaminic action. They are described by Bovet[35] as "a group of synthetic amines displaying, in most animal species, as well as in man, an antagonism specifically directed against the physiological and toxic effects of histamines." . . . "The great activity of antihistamine agents is not due to any spasmolytic, symphathomimetic or analeptic properties."

Because of chemical similarity in the amines, acetylcholine, epinephrin and histamine, and because the first two are susceptible to neutralization in the body, Bovet deduced that there might already exist substances antagonistic to the third. Some ten different chemical series were investigated, all known to have some effect on the autonomic nervous system. Six of the ten series produced drugs with definite antihistaminic action, this action being determined by:

1. Protection conferred on guinea pigs which had received several lethal doses of histamine.

2. Antagonism against histamine induced spasms of smooth muscle.

3. Protection against anaphylactic shock.

Outstanding examples of the series prototypes are: antergan from the dimethylamino group, P.B.Z. from among the aminopyridenes, and Benadryl from the benzohydrylethers. These, by no means, represent the most efficient drugs, but are mentioned purely as random examples among well-known trade names. By means of the tests outlined above, Bovet was able to predict the theoretical worth and relative efficiency of various histaminic antagonists and was able also to predict certain additional side effects on the basis of chemical structure. Later experience has confirmed these predictions and certain of these side effects have been used clinically, especially those having to do with sedation and local anesthesia.

Clinical response to the antihistamines has been excellent to poor, depending upon the type of allergy under treatment, the drug chosen, the dosage and dose interval and the presence or absence of complications. In acute nasal allergies, profuse discharge, itching and sneezing responded best and obstruction least. Acute allergic rhinitis responds better than chronic. Complicating suppurative sinusitis apparently adds a vasomotor factor which may create a turgescence completely refractory to antihistamines.

No drugs could be so potent without having some disadvantages. Among the undesirable side effects are drowsiness, vertigo, dryness of the oral mucosa, incoordination, gastrointestinal disturbances and, possibly, depression of the hematopoietic system.

In view of the wide differences in speed of action, potency, duration of effect and side reactions of different antihistamines, it should be possible to choose one best suited for almost any situation in a responsive patient.

Feinberg[36] says, "I would suggest becoming thoroughly acquainted with a group of these drugs, one of which is slightly sedative and probably less potent at the same time; a second which is potent and sedative, and a third which is potent and highly sedative." As examples of each, he suggests:

Group 1. Thephorin, P.B.Z., Decapryn.

Group 2. Neohetramine, Tagothen, Benadryl.

Group 3. Antistine, Thenylene, Chlortrimeton.

Cortiscosteroid and Adrenocortecotropin

Close on the heels of antihistamine came the works of Sprague[37] and Hench[38] with Cortisone and Adrenocorticotropin (ACTH). First offered as a control for rheumatoid arthritis, these drugs were soon under investigation by every specialty, and hopefully were tried in every condition which might conceivably be classified as a collagen derangement.

Bordley[39] was among the first to investigate the effects of ACTH and Cortisone on the nose. He compared the effects of the two drugs on normal and allergic nasal mucosae. He found that in the nonallergic patients receiving ACTH by injection the nasal mucosa becomes thinner, develops a slate pink color and is covered by a thin layer of mucus; that nasopharyngeal lymphoid tissue becomes sharply demarked, crypts become prominent and its color becomes orange pink. In the allergic polypoid nose, polyps shrink and may disappear, although there is no microscopic change in the lymphoid tissue or polyps. Sinus mucosa thins out. All these changes regress or disappear on discontinuance of treatment. Nasal polyps return in two weeks to two months. Cortisone has very much the same effect except that there is no marked color change in mucosa and no change in appearance of lymphoid tissue.

In a later paper, Bordley[40] confirms the above findings after an analysis of 500 examinations in 60 patients during and after ACTH and Cortisone therapy. After this greater experience he warns that both agents seem to lower resistance of mucosa to local infections, especially within the nasal sinuses. He feels that neither offers any hope for permanent improvement in allergies.

More fundamental investigation by Rappaport et al.[41,42] showed that nasal mucosa in allergic patients after four days' treatment with ACTH revealed:

1. A general increase in stainability of glycoprotein ground substance.

2. An increase in the cement substance between the epithelial cells of the mucosa.

3. An increase in the staining density and in the width of the submucous and of the perivascular basement membranes.

4. An increase in the number and thickness of the fibers of the ground substance and in their stainability.

5. An increase in the number and size of the red staining granules in the cytoplasm of stellate, oval and round cells present in the submucosa (believed to be undifferentiated fibroblasts and the granules to be glycoprotein in composition).

He says, "During allergic reactions glycoproteins are depolymerized. Corticotropin arrests this process and increased stainability results."

On the clinical level, there have been innumerable investigations. Being almost a pure form of nasal allergy, hay fever was the logical point of attack.

In 1951, Reicher, et al.,[43] investigated the effect of oral Cortisone (25 mg. q.i.d.) on Ragweed Hay Fever and obtained marked or complete relief in 80 per cent of their 25 hyposensitized patients. They encountered no serious side effects, although there were minor effects which did not interfere with continuance of the drug. They concluded that oral Cortisone combined with hyposensitization produced much greater relief than that obtained previously with any other treatment.

In 1952, Schiller and Lowell[44] gave as their oral dose 100 mg. Cortisone for four days to 51 hay fever patients in whom treatment with injection of pollen extracts, with antihistaminic drugs or with other agents, had been unsuccessful. There was no change in diet and no electrolyte manipulation. Complete to satisfactory relief was obtained in 42 patients. Nine obtained little or no relief. Among the 42 patients obtaining relief, 20 suffered relapse within seven days; however, only seven of these required further Cortisone. "There were no undesirable side effects."

Extending the use of the corticosteroids to perennial allergic rhinitis, Rosen and Feldman[45] reported subjective and objective improvement. Polyps became smaller and easier to remove surgically, and there was less surgical bleeding.

Weille[46] agrees with these reports, but warns that about a week after cessation of treatment, regression occurs with rebound turgescence "worse than ever and with less response to other treatment," reminiscent of similar effects of adrenergic, shrinking nose drops. He also warns that "the vasomotor rhinitis, which improves at first under systemic Cortisone treatment, but which later recurs during such treatment, is even worse."

As to side effects he quotes Bauer's report[47] of 40 patients receiving long term Cortisone treatment: three became psychotic, six developed severe infections (one fatal), two diabetics became worse, and one developed very severe hypertension. Weille further warns of possible decalcification and necessity of giving intravenous Cortisone preoperatively to patients who have been receiving the drug.

Because of its striking effectiveness in skin and superficial ocular conditions, it was logical that local applications of Cortisone and ACTH should be tried in the nose, particularly in view of the possibility of eliminating general side effects by this route.

Dill and Bolstad[48,49] sprayed Cortisone (1-4 in saline) into the nose of allergic patients every half hour for four hours and four times daily thereafter. All patients were subjectively relieved, in that nasal stuffiness and post nasal discharge were less, and in 50 per cent there was marked improvement; 24 per cent showed some, and 28 per cent showed no objective improvement. Polypi shrank but disappeared in but one case. No atrophy occurred. No microscopic change was demonstrable except reduction of eosinophiles. There were no adverse reactions.

Weille,[46] Tuft,[50] and Barger and Shaffer,[51] using Cortisone alcohol and hydrocortisone solution with and without vasoconstrictors, obtained comparable results by topical application. McKendry, et al.,[52] using ACTH by nasal insufflation,

were able to get clinical responses usually obtained after injection of ACTH. Among their 60 cases, one of allergic rhinitis was cited, in which improvement was marked and in which polyps disappeared and smell and taste returned. Other than slight nasal irritation and transient sneezing, there were no untoward effects.

In the same year Schwarz,[53] using corticotropin powder as snuff in refractory cases of allergic rhinitis, obtained complete remission of symptoms in all. (Bacitracin and tyrothricin were added with no apparent ill-result). Two cases developed allergic reactions to the material, one definitely due to corticotropin.

Herxheimer, et al.,[54] used hydrocortisone acetate as snuff in 24 cases of pollen hay fever, preceding application with shrinking agents when necessary. Results were excellent and continued good as long as the snuff was kept up. Relief persisted for as long as 16 days after discontinuing treatment and returned when treatment was resumed. There was marked concomitant improvement in ocular symptoms. There were no untoward reactions.

Evans[55] used Cortisone in combination with chlortrimeton as topical applications, sprays and packs. He had no reactions, obtained excellent results and considers the combination more effective than any previous treatment, local or general. In three cases he injected Cortisone directly into the polyps. There was marked decrease in size and no reaction.

It is known that hydrocortisone is approximately twice as effective as Cortisone, without a corresponding increase of side effects. The recently synthesized analogues of Cortisone (prednisone) and hydrocortisone (prednisolone) are much more effective still and have exceedingly few side effects. They may be given without restricting salt or giving potassium.[56] "Moon face" does occasionally occur whenever these steroids are given over long periods. As to the commonly accepted idea that the corticosteroids increase susceptibility to infection, Rawlings[57] suggests that while large, continued doses certainly do cause such a situation for reasons not ap-

parent, small doses do quite the opposite and, in fact, seem to stimulate immune responses.

The general attitude of rhinologists toward corticosteroids is best summed up by Shilkrit,[58] who says, "Corticotropin, Cortisone, hydrocortisone, prednisone and prednisolone dramatically suppress many of the clinical manifestations of allergic disease and are the most effective agents available at present for symptomatic relief in carefully selected cases, after the usual therapeutic measures have failed.

Pathogenesis

Throughout the foregoing historical review one sees that, although real progress was made in explaining the allergic state, the basic mechanisms have not yet been identified. As far as vasomotor rhinitis is concerned, it is not universally accepted that the antigen-antibody reaction is implicated at all, though this view represents an extreme and speculative attack that would seem to ignore a vast accumulation of well documented evidence.

Sheldon, et al.,[59] in 1949, say "Localized symptoms of allergic diseases . . . are the result of a systemic immunologic response to the reintroduction of a foreign protein into a previously sensitized individual . . . Evidence has accumulated to point definitely to an 'H substance,' or histamine, as the prime factor in the production of the immediate or wheal type of allergic reaction. But histamine is not the only factor at work."

In 1951, while discussing Williams'[60] paper on Autonomic Dysfunction he indicated a receptiveness to the ideas of Williams, Ashley and others, though by no means abandoning the antigen-antibody theory.

Williams does not hesitate. He says, "A theory of allergy based on the autonomic vascular reactions is felt to furnish a better working hypothesis from the standpoint of clinical diagnosis and treatment than one based on the antigen-antibody concept . . . A concept of allergy as localized or focal dysfunction is far more consistent with clinical practice than

the antigen-antibody concept; it opens up new avenues of therapeutic approach . . ."

Ashley[60] goes further. He says, "I am in active accord with these newer ideas, and believe as he (Dr. Williams) does, that all allergic reactions cannot be explained according to the antibody antigen theory of immunity. I believe it most unfortunate that the terms 'antigen,' 'antibody' and 'immunity' should ever have been applied to allergy, since it has never been shown that allergy and infections bear any relationship whatsoever.

"In an allergic reaction the normal sympathetic-parasympathetic balance is thrown out of balance, with the parasympathetic gaining dominance over the sympathetic. Extreme dilatation of the capillaries results . . . Edema is the primary cause of symptoms in all allergic reactions."

Shahinian[61] suggests that the allergy symptom complex is a local manifestation of disturbed "neurovascular physiology"; that the endocrine-hormonal system may be of importance by its action as the underlying common denominator.

Hilger[62] neatly compromises the antigen-antibody and autonomic dysfunction ideas. He says, "that hyper- and hypoactivity of endocrine or direct autonomic nervous pathways produces a disordered result evidenced by autonomic dysfunction in some end-organ. The neurovascular change is produced by dynamic alteration in the arteriolar-capillary-venular unit. The sequence of change begins with arteriolar spasm initiated by direct neural stimulation or by antigen-antibody union and injury at the smooth muscle sphincter . . ." ". . . In the nose, vasomotor rhinitis is the cardinal example of autonomic dysfunction."

In a similar vein, Woodward[63] accepts the protean origins of vasomotor rhinitis. He suggests that the terminology be revised and that antigen-antibody allergy be called "Allergy I", physical allergy be called "Allergy P" and emotional or stress allergy be called "Allergy S."

Holmes[64] says the same thing in a different way, ". . . nasal hyperfunction, as a part of an individual response to a threat-

ening life situation engendering conflict and anxiety, may constitute a major etiological factor in the genesis of nasal dysfunction. When combined with *other* environmental stimuli capable of producing nasal hyperfunction, it becomes relevant to the genesis of many acute and chronic disorders of the nasal and paranasal spaces."

It is impertinent to challenge the views of men like those quoted. It is presumptuous even to attempt to compromise them, but one must try to sort their views into a working hypothesis for his own guidance.

If it is assumed that an "H substance" (histamine) is the end-product of antigen-antibody reaction, whether produced by protein allergens or physical exposure, and if one can accept as a fact that the "H substance" is elaborated in emotional stress or as the result of "stress" or "alarm reaction" or "adaptation syndrome," it is not reasonable to assume that each can result in vasomotor imbalance, which is but an intermediate step toward the edema which seems accepted as the true common denominator?

To separate these various cases of vasomotor rhinitis into pure and unmixed forms is impossible. For instance, it may be that in the first isolated vasomotor response there is no secondary infection, but that after repeated acute episodes or persistent perennial reaction, infection in some degree results. Whether infection acts as a true allergen or as another of the stress factors that encourage an unstable vasomotor system is a question, but there is no question whatever about its ability to make an existing allergic situation worse and to prolong its course. In every individual there is constant stress, constant emotional stimulaiton and never-ending adjustment to physical insults. Anderson, et al.,[65] have resolved the ensuing situation into a very plausible theory which they call the theory of Allergic Load, and which they define as the sum total of the factors which play a part in the production or modification of allergy symptoms in the allergic person, these factors being "contributory and exciting." They say, "The contributory factors are incapable of producing an allergic attack by themselves, but they affect the person in such a way as to precipitate or aggravate allergic reactions.

In many instances they act by adversely affecting capillary function. They are: mechanical irritants such as dusts, chemical fumes and pungent odors . . . infection, emotion, changes in weather, barometric changes, endocrine unbalance, physical and mental exhaustion, malnutrition, changes in mineral and lipid metabolism, etc. Specific or exciting factors are the antigens or allergens. They suggest that each allergic patient is in a constant state of potential reaction and that actual reaction may be the result of any adequate combination of exciting factors.

PATHOLOGY.

Whether due to antigen-antibody reaction or to "autonomic unbalance" the basic pathology in vasomotor rhinitis remains the same, and the immediate and late effects of edema represent the essential pathology of both.

Under proper stimulus, a constant phenomenon occurs, and is a part of every atopic nasal reaction: spasm of arteries and veins, dilatation of venules, increased capillary permeability and stasis. In the nose, the ultimate pathology seems to be a breakdown of the cementing ground substance between the collagen fibrils[66] in the tunica propria, with accumulation of fluid in the spaces thus created. This same edema affects epithelium as well and results not only in swelling but also in decreased gland and ciliary activity. If the stimulus and its consequent reaction is severe enough, sloughing of surface epithelial cells with their cilia may result. Along with the actual intra- and extracellular edema there is a parasympathetic hyperactivity which results in generally increased vascularity and distention of blood spaces in the erectile tissue of the turbinates and septum. As far as the nose is concerned, this is a total reaction, all areas of the nose and sinuses participating to the degree permitted by their histological anatomies. It is also total in the sense that the entire thickness of the mucous membrane, even periosteum, is involved; with general engorgement, intra- and extracellular edema, swollen and hyperactive glands, infiltration of tunica propria with lymphocytes heavily interspersed with eosinophilic leucocytes, and desquamation of epithelium mixed with

extruding eosinophiles. In its simplest form this process is represented by isolated reaction to evanescent stimuli (as in late seasonal pollen allergy) and is probably completely reversible. Too frequently repeated reactions or more constantly and persistently applied (perennial) stimuli will result in irreversible change in which we see varying degrees of:

1. Epithelial metaplasia and diminution of cilia.

2. Stratification of epithelium.

3. Increase in goblet cells.

4. A significant change in composition of normal surface mucus in which are suspended epithelial cells and characteristic eosinophilic leucocytes.

5. Myxomatous appearance of stroma cells with many young fibroblasts, round cells and eosinophilic leucocytes.

6. Cyst formation.

7. Widening of tissue spaces (these spaces filled with tissue fluid in which there tends to be an electrolyte shift from potassium to sodium.

8. Nutritional or pressure changes in supporting bone.

Possibly with all the above findings present, there is still a point at which the process is completely reversible; but, exposures being what they are, the tendency is usually toward the irreversible or degenerative stage in which all of the above findings are increasingly present and in which gross edema results in polypoid degeneration. By their very weight and loss of normal "reactability" the affected membranes sag and form all degrees of polyps.

Hansel[67] describes the gross appearance of chronic nasal allergy: "In typical cases, the mucosa appears pale, greyish in color, boggy or swollen. Occasionally it appears normal, or somewhat red. Pale, edematous swelling may be noted in the middle meatus. Definite polyposis may be present. It is almost always bilateral, but may be unequal in degree. Edematous polyps do not form on the septum or the inferior turbinates. Allergic papillary growths may be noted occa-

sionally on the septum and the inferior turbinates or in the vestibule of the nose. Mulberry hypertrophy of the posterior tips of the inferior turbinates may be noted . . . Allergic polyps are rare in children before puberty."

Secretions present vary in appearance from clear and but slightly viscid to gelatinous, and seem more copious in the posterior than in the anterior nose. To these findings may be added the characteristic changes due to infection, in which the secretions may be more purulent and in which one or more sinuses may be the seat of retained pus and mucus. This is the usual gross appearance of perennial allergic vasomotor rhinitis.

ETIOLOGY.

Although in infancy, food allergens are the usual causes of allergic reactions and the skin and gastro-intestinal tract the usual sites, after age 3, inhalants seem to be the important excitants and the nose and bronchi the shock organs. As a consequence, allergic rhinitis is rare in early childhood, becoming increasingly common through adolescence, and reaching its greatest incidence in middle age. Sex seems to have no bearing except perhaps in the menopause.[68] Heredity certainly is a factor, though not a direct one. Body type, color and personality may have some influence, but their implication has not been proved. Occupational, seasonal and geographical influences obviously are important as are emotional upsets, either as "stress factors" or as specific excitants. Whether diet, except in its specific action, is important, is questionable although proper intake of vitamins, of salt, and proper balance of all nutritional elements, would seem essential to proper body function.

Although, as mentioned, food allergy is not common in allergic rhinitis, it does occur, especially in company with inhalant sensitivities. Among the common food allergens are wheat, egg, milk, fish, nuts, beans and peas, potato, tomato, onion, celery, cocoa and yeast.[69]

In asthma, the epidermal allergens are important, but with the exception of feathers, they seem to have little connection

with allergic rhinitis. Fungi, especially Alterneria, Homodendrum and Penicillium are frequent offenders, and with the tree, grass and weed pollens account for most seasonal nasal allergies.

Cosmetics, particularly those containing orris root; wave set, cotton seed, kapok and a host of other substances common to households may be significant. Among the latter, and certainly the outstanding offender in perennial allergic vasomotor rhinitis, is house dust, which according to Feinberg,[69] is not simply a mixture of common respiratory allergens but, rather, is a substance formed in the house from disintegrating vegetable fibers such as cotton, and tending to accumulate in such areas of maximum proximity as mattresses, bedsteads, etc.

Physical factors such as heat and cold can act as true allergens. In connection with nasal allergy they more probably are exciting factors of a secondary nature, though none the less important for this reason. Many individuals in precarious vasomotor balance because of existing perennial allergies, studiously avoid drafts and chilling, because of their certain foreknowledge of the explosive result.

Infection, especially of the sinuses, is probably not a primary cause of vasomotor rhinitis, but as a secondary factor it is not only important but in many cases becomes more so than the original allergy, because of its debilitating effect on the patient. Many cases of pure seasonal allergic rhinitis will carry symptoms indefinitely until relieved of a complicating empyema of a maxillary sinus. Acute exacerbations in perennial cases often occur on the basis of ethmoidal and maxillary retention of surprisingly small amounts of mucopus (perhaps this is still another type of secondary stimulus on a foreign body basis). Eventually, most cases of allergic vasomotor rhinitis, unless treated vigorously and adequately, will reach a stage where surgery for the control of infection becomes imperative. This marked tendency toward infection is fostered by such allergic effects as ciliary damage and change in composition and quantity of mucus and, of course,

by blocking due to edematous mucosa and polyps. Existing anatomical obstructions are of the greatest importance.

SYMPTOMS AND DIAGNOSIS.

There should never be difficulty in identifying an acute allergic nasal reaction except one due to nonspecific, direct irritation or an occasional drug reaction such as the turgescence caused by iodides and rauwolfia. The extreme blocking, itching, rhinorrhea and lacrymation are so characteristic as to need no further description. Occasionally headache and sore throat, as the early symptoms of an allergic episode, are confusing. Nor should an examiner be in doubt as to the cause of chronic obstruction, when typical pale mucosa and diffuse polyposis is present, though most of our patients are not so considerate as to have such typical and uncomplicated symptoms and findings.

Headache, diffuse or limited sharply to sinus areas; obstruction, loss of taste and smell, postnasal secretion, crusting, nasal voice and "baggy" eyes, along with a nagging desire to clear the nose, are complaints common to most chronic nasal conditions. If itching of nose and conjunctivae are present and if paroxysms of sneezing occur an allergic element must be strongly suspected.

History is all important in differential diagnosis. A chronic nasal condition which dates directly and positively from a severe respiratory infection, extraction of an upper tooth, trauma to the face, diving, etc., is almost certainly infectious in origin. On the other hand, one which began after a change in environment, and is worse in certain environments or seasons, or perhaps is accompanied by food intolerances, skin diseases, or asthma, or has been present "as long as I can remember," usually has a heavy vasomotor component.

Inspection of the nose is not always completely informative, although the typical boggy, grey membrane, when present and when accompanied by polyps, is highly suggestive of allergy. Secretions are deceptive. While not the usual picture, heavy opaque secretions can be present in the allergic nose with a minimum of infection, whereas in frank empyema

of a maxillary sinus the nasal secretions may be clear and watery and obstructive edema marked. The presence of eosinophiles is significant, as shown by Hansel[70] and others. As a generality, the larger the proportion of eosinophiles in the nasal smear, the greater the allergic factor. Absence of eosinophiles in nasal secretion does not absolutely exclude allergy, as occasionally, cytological examination of material recovered from sinus washings in the same patient may show a significant number of eosinophiles, as may the differential blood count, particularly if foods are the allergens responsible.

X-ray studies of the nasal sinuses can be at the same time, helpful and confusing. Thickening of a single sinus mucosa or of sinuses on one side suggest an infectious process. Marginal thickening of the mucosa of many sinus areas, especially, if from exposure to exposure, there is wide fluctuation in the degree of involvement, is pathognomonic of allergy. Softening or absorption of sinal bony outlines is considered diagnostic by Hansel.[70] Installation of radiopaque solution either by direct injection or by Proetz displacement is invaluable in X-ray interpretation. Transillumination, notoriously unreliable, does give valuable information, nevertheless.

Antrum puncture will make contained mucus or mucopus available for cytological and bacteriological study. Antroscopy can reveal polypoid membranes not otherwise visible and nasopharyngoscopy will often show polyps which would not have been discovered by any other means. Finally, the differential blood count sometimes reveals an important eosinophilia, and observation of the "leucopenic index," as reported by Rinkle,[71] can be helpful.

Before a diagnosis of *allergic* vasomotor rhinitis can be justified, it would seem necessary to consider the possible causal or contributing effect of physical exposure, nonspecific irritants, emotional influences and environmental stresses; but once established, such a diagnosis calls for pursuit of the offending allergen or allergens.

Here history is all important. In a high proportion of cases, a careful, exhaustive interrogation will bring out seasonal and environmental characteristics which will identify

the pollens, dust, cosmetics, etc., which are implicated. Information thus obtained will be corroborated in skin tests with the suspected material.

Whether the degree of sensitivity can be related to the size of the wheal obtained in skin testing is a question. Feinberg says, "my experience indicates that, on the average, a large skin reaction indicates a greater degree of clinical sensitivity than a small reaction." Rinkle believes that, as an index for treatment, wheal size is not nearly so important as titration.

When history and skin tests do not solve the diagnosis, simple eliminations and environmental "cleanup" may help. Response to antihistamine is sometimes spectacular and, therefore, broadly diagnostic, but lack of response cannot be relied upon as a negative test.

TREATMENT.

There is probably no disease involving so limited an area in which treatment of the "whole man" is more necessary than in allergic vasomotor rhinitis. Before any definitive treatment of the nose can be considered, all the factors of environment, physical and psychic, must be evaluated and adjusted, due consideration being given to the fact that radical and inept attempts at such adjustment may leave the patient worse off than before. Strict attention must be paid to the patient's own estimate of the source of his trouble, for the clues thus gained may be the basis for successful treatment, and the time expended a capital investment in good doctor-patient relationship. Before any plan is begun, there should be, between doctor and patient, an absolute understanding of the purpose and limitations of whatever treatment is undertaken. The patient must know at the outset that treatment limited to relief of obstruction is temporary, whether the relief is gained through polypectomy, elimination of anatomical deformities or electro surgical or chemical means; that such treatment (which is all most patients seem to want at first) is to be discouraged; that truly adequate care requires cooperation in a complex and sometimes

protracted program which ultimately should be worthwhile, but which may involve backsets and disappointments.

Acute cases, and those whose duration has not been long enough to result in marked hyperplasia and polyposis should be more easily and quickly reversible. Most of these are of pollen or mold origin and respond well to antihistamin, with an occasional assist from steroids. Surgery is rarely necessary, and usually consists of no more than adequate drainage of a pus-filled maxillary sinus. Identification of the offending allergen is usually not difficult. Eliminations and environmental cleanup may effect a cure, though, in the case of pollens and molds, preseasonal hyposensitization is usually needed.

Unfortunately, most of the patients with chronic allergic vasomotor rhinitis present themselves after months and years of self treatment, inadequate or ill-chosen treatment, or no treatment at all. Many have had nasal surgery and are in no mood for more, just as many have undergone hyposensitizing injections with no relief and sometimes with violent reactions. Most have complicating infections. The situations of some of them might seem hopeless were it not for the possibility of multiple attack which has now evolved.

Although each patient is different from the last, a systematic routine can be applied, there being a heavier emphasis at different points of the routine in different individuals. An outline of available approach follows:

General Treatment—Careful history leading to:

Stress evaluation and adjustment.

Environmental cleanup—allergen free bedroom, etc.

Elimination of suspicious specific factors—physical, ingestant, inhalant.

Establishment of basically adequate diet high in vitamins, low in sodium.

Hyposensitization with allergens, histamine, toxoids.

Drug Therapy—Antihistamines.

Steroids.

Antibiotics in subacute and surgical phases.

Local Treatment—Non-surgical.

Nasal vasoconstrictors.

Injection of sphenopalatine ganglion.

Semipermanent reduction in turbinate size.

Chemicals-cauterization, sclerosis; electric-ionization, diathermy; escherotic-electrocautery, submucus resection.

Radiation—Radium to polyps.

X-radiation of sinus areas.

Surgical—Correction anatomical deformities.

Polypectomy.

Exenteration sinuses.

Tonsillectomy and adenoidectomy.

At the outset an immediate problem arises. Should the rhinologist attempt to carry the whole burden of the patient's care, or should he enlist the aid of the internist, the allergist and, perhaps, the psychologist? The answer depends upon the availability of such specialists, the degree of cooperation obtainable and the rhinologist's estimate of his own ability to handle these collateral factors with more good than harm to the patient.

Our own routine involves a complete physical examination by an internist. When allergic causes are not quickly discovered by question, and by inference and elimination; and for all skin testing, our patients are referred to the allergist. We have failed to attack psychosomatic causes with vigor because of skepticism as to our own ability to evaluate and correct such factors, and because of instances in which resistance and resentment have met our suggestion of referral to a psychiatrist.

Our definitive treatment lies somewhere between an adamantly non-surgical approach and the "pan-sinusectomy" of earlier days. Where allergens other than dust are unequivocally involved, all means are used to eliminate them from the patient's environment, to remove the patient from exposure and/or to maintain hyposensitization. Dust sensitive, and those whose allergic background is in doubt, receive stock dust

injections in dilutions low enough to give a minimal local redness with *no* general reaction. If nasal stuffiness occurs and persists, the subsequent dosage is reduced until no nasal reaction occurs, then is gradually built up again. Beginning with subcutaneous injections twice weekly, the interval is gradually increased until 14 days is reached, at which interval treatment is maintained. In general, the skin reaction to intradermal testing with *the extract to be used therapeutically* is followed as a guide to dosage, the intradermal test being repeated, from time to time, as a check against sensitivity.

Antihistamines, following the suggestion of Feinberg[36] as to choice, are started early. Nasal decongestants are permitted, but their frequent use is discouraged, and the patient is asked to eliminate them as quickly as possible "to prove that his treatment is taking effect." Very early attempt is made to minimize the effect of complicating infection by antrum irrigation, removal of polyps blocking the middle meatus, and by the use of antibiotics.

At this point definite improvement may be apparent, and the program is maintained at this level. If no improvement is evident and if polyposis is not too much a factor, more aggressive means to reduce obstruction are used. Prednisone is given in amounts varying from 30 to 60 mg. per day for two days. The dose is reduced sharply on the third day to from 15 to 25 mg. daily for from one to three weeks. After the third week our use of prednisone varies, depending upon nasal response and upon unwanted side effects (these are few and usually completely absent). We do not give steroids to any patient with gastric ulcer or severe infection, nor do we perscribe it in patients who are obviously disturbed mentally. Our results with topical steroids have not been brilliant. Occasionally, subjective and objective results are spectacular and are maintained as long as optimum doses of steroids are given; however, the drug is not a cure-all, is too expensive for long time use by many patients and is not a substitute for more definitive treatment.

Other means short of sinus surgery may be used to reduce nasal obstruction. Intranasal polypectomy, shrinkage of

polyps and turbinates by injection of sclerosing agents, diathermy, chemical application and cautery, are all used in selected cases.

Our results from radiation have been disappointing. No demonstrable change in hyperplasia has occurred and reactions have sometimes been violent.

Having had to cope with the end-results of the mass "slaughter of noses" that occurred in the early part of this Century, the author at first shared the resulting widespread skepticism as to the need for most sinus surgery; but experience with advanced vasomotor rhinitis of the allergic type has proved that properly planned and executed nasal surgery is often the essential step, without which the patient will not improve[72] in spite of vigorous anti-allergic and decongestive therapy.

Submucous resection, antral and ethmoidal exenteration with preservation of the middle turbinates where possible, and removal of polyps at their sources are performed when necessary, with expectation of real benefit to the patient, and with no apprehension as to mutilation or interference with function. The identification and removal of an occasional malignant degeneration further justifies such surgery.

In spite of recurrence of polyps and occasional need for further surgery, most patients treated in this fashion have been kept decidedly comfortable. They do tire of the never-ending "shots", and too many stray away, but many can be classified as "much improved" on the program, and show every intention of continuing treatment.

Following are four case records, all patients with allergic vasomotor rhinitis with varying degrees of hyperplasia and secondary infection:

R.G.C., white female aet 65, housewife—November, 1952. This patient had had asthma for ten years and suffered from almost complete nasal obstruction. There was marked thickening and retraction of both drum membranes. On transillumination the left maxillary sinus was dim. Antrum puncture obtained mucopus, 5 c.c. from the right, 10 c.c. from the left, which showed pure culture of alcaligenes and no eosinophiles.

X-ray showed marked thickening in left antrum and moderate in right, and the nose "almost completely occluded by increased soft tissue density."

Skin tests: Many foods positive. House dust 1+.

Right anterior and posterior ethmoidectomy and polypectomy was followed in 24 hours with left ethmoidectomy and polypectomy, patient was placed on diet, dust injections and chlortrimeton. Because of subacute flareup in right maxillary sinus, a right antral-meatal opening was made in January, 1953. She showed only moderate symptomatic improvement and continued to form ethmoid polyps until April of 1953, when hydrocortisone 50 mg. q.i.d. was given and reduced to 25 mg. t.i.d. after two days. Dust injections and chlortrimeton were continued.

Since this time she has received steroid therapy one week out of three with marked subjective and objective improvement, except for moderate persistent formation of ethmoid polyps.

R.C.B.., white female, aet 45, housewife—January, 1953. Patient had had asthma for years and had been told she had secondary bronchiectasis. She had marked bilateral nasal obstruction. She had had no nasal surgery and no treatment with steroids previous to admission.

X-ray of chest showed a bronchiectasis. X-ray of sinuses showed marked pan sinusitis. The nose showed pale, boggy mucosa, polypoid middle turbinates and obstructive polyps bilateral. Antrum irrigation produced clear mucoid globules, smears of which showed no eosinophiles.

Allergy study showed sensitivity to feathers, housedust, "mold" extract and mill dust.

On January 27, 1953, right ethmoidectomy and polypectomy were done, and left ethmoidectomy and polypectomy two days later. Patient was placed on dust injections and chlortrimeton, but by April, 1954, was again developing obstructive polyps, which were removed. Polyps again slowly recurred, and on May 2, 1955, patient was started on meticorten 5 mg. q.i.d. for two days. This dose was reduced to 5 mg. t.i.d. and continued at this level until May 16th. At this time the polyps had receded markedly. By July 28, 1955, the polyps were no longer visible. Dust and chlortrimeton were continued and meticorten 5 mg. t.i.d. was given one week on and two weeks off. When last seen in July, 1956, the patient had no nasal complaints and no polyps were visible. There was, however, some polypoid change of both middle turbinates.

J.T., white male, aet 56, physician—January, 1954. Chief complaint—marked nasal obstruction. Previous history—frequent, persistent head colds, sometimes complicated by "wheezy" cough. Severe contact dermatitis during World War II during service as Naval Surgeon. Dermatitis found to be due to alcohol, mercury, sulfonamides and hyposulphate solution.

In January, 1953, usual "winter cold" became steadily worse until nasal obstruction and severe wheezing attacks became incapacitating. Patient obtained no relief from antihistamines and repeated antrum washings. X-ray treatment caused violent reactions, and obstruction was not relieved. Antibiotics were of no help.

Physical examination—typical boggy nasal mucosa, entire nose, polypoid degeneration both middle turbinates, small polyps in middle meatuses. Profuse clear secretion. Both antra dark to transillumination. X-ray-marked hyperplasia of lining membranes of maxillary and ethmoid sinuses confirmed by lipiodol studies.

Antrum irrigations revealed drams I of mucopus in each. Nasal smears varied from 12 to 30 per cent eosinophiles. Except for mild asthmatic bronchitis patient's general examination was normal, and skin tests were inconclusive, there being a slight positive reaction to house dust.

Patient was placed on a regime of house dust injections, thephorin t.i.d. and hydrocortisone 25 mg. t.i.d. He obtained relief as long as hydrocortisone was continued, but the nose was basically unimproved until a bilateral ethmoidectomy was performed in January, 1955. Since this date there has been no recurrence of symptoms.

Present treatment consists of house dust injection every second week and copyronil once daily. No steroids have been taken for six months. The nose is clean, the mucosa is pink and X-ray of sinuses shows a remarkably good aeration and a minimum of mucosal thickening.

R.D., white male, aet 45, executive—May, 1953. Patient has been asthmatic for four years and has had no overall improvement on dust injections, antihistamines and bilateral polypectomies. He is known to be allergic to alcohol.

Examination showed a typical pale, wet, boggy nasal mucosa and marked bilateral polyposis. Antral punctures revealed .5 c.c. organized flakes in each, from which there was no growth on culture.

X-ray sinuses—reported "advanced pan sinusitis." Blood count was within normal limits except for an 8 per cent eosinophilia. Skin testing revealed a mild reaction to mill dust and moderate reaction to combined house dust.

Bilateral ethmoidectomy and polypectomy were performed, and followed in three months by a submucus resection. In the meantime dust injections were begun. *Two* months later there was a return of polyps and a bilateral maxillary sinusitis, purulent—relieved by irrigation and removal of ethmoid polyps. Patient was continued on dust, and chlortrimeton and hydrocortisone were begun. After six months there was no return of symptoms and no ill effects from hydrocortisone. The program was modified to continued dust injections, chlortrimeton twice daily and hydrocortisone one week out of each three months. At his last examination in June, 1956, patient was symptom free and the nose was in "good condition."

SUMMARY.

A survey of the history of the advances in allergy has been presented, with detailed discussions of the more important factors as they apply to allergic vasomotor rhinitis. Etiology, pathology and pathogenesis, with an outline of available treatments, precede a statement of the author's treatment routine and a presentation of typical case reports.

CONCLUSION.

Allergic vasomotor rhinitis, though rarely cured, is not a hopeless disease. It can be controlled to the point of patient satisfaction, by a systematic and carefully planned routine, varied as to the individual's needs, but aimed at all these needs. Hyposensitization, control of edema with steroids and proper surgery form the basis for that routine.

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SEVENTH INTERNATIONAL CANCER CONGRESS—LONDON, ENGLAND.

The Seventh International Cancer Congress will be held in London, England, July 6-12, 1958, under the Presidency of Sir Stanford Cade. Congress headquarters will be The Royal Festival Hall. This Congress is sponsored by the International Union Against Cancer.

There will be two main sessions of the Congress: A. Experimental; B. Clinical and Cancer Control. Special emphasis ill be placed on hormones and cancer, chemotherapy, carcinogenesis and cancer of the lung.

Papers will be considered only if submitted with an accompanying abstract (not over 200 words) before October, 1957, and if dealing with new and unpublished work.

The registration fee for the Congress will be £10 (ten pounds) or $30, and the latest date for registration without late fee will be January 1, 1958.

Registration forms and a preliminary program will be available early in 1957 on application to The Secretary General, Seventh International Cancer Congress, 45 Lincoln's Inn Fields, London, W. C. 2, England.

AMERICAN MEDICAL ASSOCIATION PROGRAM FOR THE SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

New York, June 3-7, 1957, Ballroom of the Park Sheraton Hotel.

Tuesday, June 4, 1957:

Honorary Chairman's Address—Dr. Julius Lempert.

Two case reports (five minutes each).

"Elongated Styloid Process"—Guy Owsley, M.D., Hartford City, Indiana.

"New Tracheotomy Tube"—Ralph Caparosa, M.D., Pittsburgh, Pennsylvania.

"Sudden Deafness of Obscure Origin"—Eric Hallberg, M. D., Rochester, Minnesota.

"Monocystic Fibrous Dysplasia of the Maxillary Sinuses"—Jose Pico, M.D., Avenue Ponce de Leon 654, Santurce, P.R.

"Symposium on Virus Disease of Respiratory Tract, Diagnosis, Chemical, Laboratory Treatment, Etc."—Dr. Gordon Hoople, Moderator.

"Laboratory Side of Virus Disease, Etc."—S. S. Kalter, Ph.D. (guest), Montgomery 1, Alabama.

"Diagnosis of Virus Disease, Etc."—Albert P. McKee, M. D. (guest), Iowa City, Iowa.

"Chemical Side of Virus Disease, Etc."—Harry Schenk, M.D., Philadelphia, Pennsylvania.

Wednesday, June 5, 1957:

Two case reports to be given.

"Correction of the Nasal Tip in Rhinoplasty"—Sam Bloom, M.D., New York City 28, New York.

"Nasal and Laryngeal Involvement in Abdominal Hodgkin's Disease"—Herbert A. Lautz, M.D., Hammond, Indiana.

"The Full Ear"—Victor Alfaro, M.D., Washington, D. C.

Chairman's address by Gordon Hoople, M.D.

"Symposium on Headache"—Henry Ogden, M.D., New Orleans, Louisiana; Stanley Batkin, M.D., Syracuse, New York; and Raymond L. Hilsinger, M.D., Cincinnati 2, Ohio.

Thursday, June 6, 1957:

Two case reports.

"Diseases of Salivary Gland"—Robert Martin, M.D., Zanesville, Ohio.

"Tumors of Sinuses and Orbit"—Gordon Castaglino, M.D., Philadelphia 4, Pennsylvania.

"Symposium on Therapy in Otolaryngology."

"Use of Corteo Steroids"—Louis E. Silcox, M.D., Philadelphia 31, Pennsylvania.

"General Therapy in Otolaryngology"—Herman Z. Semenov, M.D., Beverly Hills, California.

UNIVERSITY OF ILLINOIS.

The next course in Laryngology and Bronchoesophagology to be given by the University of Illinois College of Medicine is scheduled for November 4-16, 1957, under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

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Annual Meeting: Palmer House, Chicago, Ill., October, 1957.

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Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

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Secretary of Otolaryngology Section: Dr. Robert W. Godwin.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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